PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

J. Y. W. MacALISTER

UNDER THE DIRECTION OF

THE EDITORIAL COMMITTEE

VOLUME THE TENTH

SESSION 1916-17

PART III

SECTIONS:—

ODONTOLOGY OPHTHALMOLOGY OTOLOGY PATHOLOGY
PSYCHIATRY SURGERY THERAPEUTICS AND PHARMACOLOGY

NOTES ON NEW BOOKS

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LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1917

LONDON:
JOHN BALE, SONS AND DANIELSSON, LTD.,
OXFORD HOUSE,
83-91, GREAT TITCHFIELD STREET, OXFORD STREET, W. 1.

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SECTION OF ODONTOLOGY

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SECTION OF ODONTOLOGY.

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Section of Odontology.

President — Mr. W. B. Paterson, F.R.C.S., L.D.S.Eng.

(*November* 27, 1916.)

PRESIDENTIAL ADDRESS.

By W. B. Paterson, F.R.C.S., L.D.S.Eng.

I thank you for the honour you have done me in electing me your President. It is an honour which I greatly appreciate, and shall strive to deserve. We meet under circumstances of an unparelleled kind. The War's demands upon the services of dentists, who are of military age, have robbed this Section of the Royal Society of Medicine of its chief interest, for to attend a dental meeting nowadays for any other purpose than war business, is like a performance of Hamlet would be with the name part cut out. We must however carry on such business as arises, notwithstanding diminished interests and attendance of members. Many of us, who are past the fighting age, regret to see our dental brethren called up to serve as combatants in the ranks, at a time too, when there is a shortage of Army doctors. I say nothing of dentists for dentistry, nor of the formation of a Dental Corps for the Army and Navy. Such matters must stand over in view of the Government's pronouncement that the present system is satisfactory, and that "the supply of dentists for the forces is adequate." But I cannot help feeling that if only the War Office authorities would recognize the wisdom of calling dentists back from the ranks, and would employ them in their medical service, they could, at once, obtain valuable surgical assistance, such as dentists, by manual training and hospital experience, are able and willing to give. This, however,

is not the place, even if it were the time, to discuss such questions; and moreover, the British Dental Association may safely be trusted to watch over them. I shall therefore, without further comment, suppress my strong personal feelings, and proceed with the business of the evening.

In the first place I have to express my regret that I have found it quite impossible to concentrate my mind, during these troublous times, upon a subject of purely scientific interest, which might fulfil all the traditional requirements of a Presidential Address. If therefore the few, somewhat desultory remarks that I venture to make by way of apology for such an address fail to interest you, I must plead the all absorbing topic of the War as my defence, and beg that you will extend to me your kind indulgence.

From all I have seen and read of jaw injuries in this War, and from the limited number of gunshot cases that I have personally treated, I should imagine that the time is not ripe yet to express a final judgment upon the various methods of treatment which have been adopted or advocated. Those of us who have had much experience of general hospital practice have seen and treated many jaw injuries, and possibly our treatment of some of them, as in the case of fractures, may have become somewhat stereotyped. But all of us, I think, must admit that the results of gunshot injury in this War have very considerably altered our methods of treatment. More especially is this so in cases of fracture of the jaw. In these cases we see great comminution of bone, splintering of teeth, extensive laceration of soft tissues, and the common supervention of sepsis. And these complications in conjunction serve to modify greatly the methods of treatment formerly pursued in civil practice. For an example let me cite the use of mouth splints. In civil practice it is, and always has been, the aim of the dental surgeon to immobilize a fracture of the jaw by an interdental splint, at the earliest possible moment, thereby converting a compound fracture, as it is, with attendant risks of suppuration, into a simple fracture; and by the immobility obtained allowing rapid healing of the torn soft tissues, as a protection against sepsis. Now, owing to the severe and smashing nature of the injury to both hard and soft tissues, and the association of septic conditions in the wound from the outset, treatment by immediate splinting, interdental, or otherwise, is necessarily delayed until such time as the septic inflammation is either past, or greatly allayed. In some cases the swelling and sloughing of the wound is so extensive as to prevent the possibility

of any form of interdental splinting being applied at all. I have seen more than one such case of multiple fracture recover, with complete restoration of masticatory function, under no other treatment throughout than free drainage and antiseptic lavage of the wound, combined with the external support of gauze padding and bandage—a lesson, it may be said, to any dental surgeon in a hurry to apply mechanical treatment, in Nature's powers of repair if unimpeded.

For another example of the differences in treatment, as seen in civil and war practice, I turn to the forms of mouth splints in use. The Hammond splint, a wire interdental splint, much in favour in the treatment of fractures of the body of the mandible in civil practice, is now much less used in war practice. Tooth capping plates cemented on the teeth appear to have superseded it, as both simpler to make and easier to apply. Although the risk of altering the "bite" by a capping plate is ever present, it may be regarded as almost negligible when care is exercised.

The Gunning splint of single, rather than double, form, either made of vulcanite or metal, appears to keep its place in war practice, as in civil practice, as a most useful means of immobilizing fractures of both jaws. Its tendency to hold food and impede the free flow of discharges does not appear to promote sepsis to any but a very slight extent, owing no doubt to the greater care that is now taken in the nursing of the case by means of frequent antiseptic douches. Even the unpleasant odour of vulcanite in the presence of suppuration, or the still worse odour of the silver, or alloy splints, is readily overcome by douchings and deodorants.

In the many and various devices for splinting fractures as seen in this War "necessity is the mother of invention." Besides the splints of civil practice like the Hammond, the Gunning, and the Kingsley, other forms of fixation have been borrowed from orthodontic practice and dental mechanics generally. In fact there are no limits to the varied ingenuity that a skilful dentist may display in his work. One form of fixation of fracture of the mandible—viz., wiring the bone—does not appear to have found favour with surgeons or dental surgeons in this War, doubtless owing to the septic nature and greater comminution of gunshot fractures. In civil practice in the past, jaw-bone wiring had few advocates at best. I confess I was never one of them.

To take another example in a comparison of civil and war practice. A prominent feature in war practice is the care taken to save every bony fragment in a multiple and comminuted jaw fracture, except

such as is obviously in a state of necrosis or sequestration. It was formerly asserted that the mandible was more prone to necrosis in comminuted fracture than the maxilla, owing to an alleged poorer blood supply. This War has shown us cases of multiple fractures of the mandible uniting most satisfactorily. I recall at the moment a case in which the mandible showed in the radiograph five lines of fracture at the angle. Any one of the loose fragments could easily have been removed, and doubtless would have been removed some years ago, in order to prevent the onset of probable necrosis. Nevertheless the parts became adherent, and the angle was perfectly restored both as regards appearance and function some four months afterwards.

Whilst war practice has taught us to save bone fragments, it has dealt hardly with the teeth. Many teeth, both near to and remote from the lines of bone fracture, have had to be removed to obviate delay in bony union, some as a result of fracture in themselves, others of loosening, and others again which were the subjects of septic pulp or periodontal inflammation.

Differences of opinion among dental surgeons prevail as to the question of septic teeth. Some limit the operation of extraction to the teeth immediately adjacent to the fracture; others freely extract all teeth to which the term "septic" can be applied. In civil practice we must often have seen fractures of the maxilla and mandible unite, within an average period of from six to eight weeks, where no teeth, either clean or septic, have been removed. In cases of delayed union we have commonly found a loose tooth, or a root in the line of fracture, to be the cause. Furthermore, we have probably noticed that the extraction of the tooth on either side of the line of fracture, in a crowded dental arch, diminished the risk of delayed union from loose teeth, and prevented the after-formation of a gum pocket. But war practice has modified one's attitude towards the extraction of teeth. Our Curator, Mr. J. F. Colyer, has lately demonstrated to us certain cases of delayed union of fractures caused by septic teeth. Teeth, apparently remote from the line of fracture, were found to be directly in connexion with it by means of fissures running through the bone. This condition was quite unsuspected and could only be observed in a radiograph. In future, therefore, we must pay more attention to the position of septic teeth in relation to the fracture, and our chief assistance will be found in a clear and well-taken radiograph.

Before leaving the subject of fractures, I should like to institute one more comparison between civil and war practice, in a reference to frac-

ture of the ramus. Here I venture to think that the treatment for once coincides. In civil practice I have never found it practicable, or even necessary, to apply any form of internal mouth splint for fracture of the ramus. I have seen excellent results follow upon treatment by external support only, such as pad, bandage, and a metal chin splint of aluminium or tin, which can either be made by the dentist or procured from the surgical instrument maker. In war practice I have seen similar good results follow upon the same treatment.

Another class of injury, but seldom seen in civil practice although quite common in war, is that in which a considerable portion of the body of the mandible is lost. We are all aware of the ghastly deformity which so frequently results from the ends of the bone falling together, and uniting either by fibrous tissue or bone. We recognize the loss of all dental occlusion, as well as the impossibility of restoring it by mechanical means. Most of the models shown at our recent meetings illustrate the belated attempts made by dental surgeons to prevent permanent deformity, by expanding the contracted arch before complete osseous union has occurred. We dental surgeons are all agreed that the sooner we can be called in to treat such cases the better is the outlook for the patient. I need not trouble you with the details of treatment, as both principles and details are well known to you. What I desire to refer to more particularly, in these cases of considerable loss of bone, is the question of the patient's future. In one case we see the mandibular arch contracted and deformed to such an extent that any dental occlusion is impossible. In another case we see the mandible in two portions both freely movable upon their condylar joints, and upon one another, the free ends remaining connected by a fibrous band. Both conditions mean for the sufferer a miserable existence, and a minced diet for life. There is a chance of improvement in the second case, or that of fibrous union, provided sufficient jaw rigidity can be obtained by a bridge fixed to the teeth. In the absence of conditions favourable for bridgework, we are obliged to have recourse to a form of removable denture which spans the space between the bone ends by fitting into it. Not a satisfactory arrangement for masticating anything beyond soft food, and an extremely irksome apparatus to wear when one portion of the mandible contains no teeth to which it can be fitted or capped. I have had examples of both these cases under my observation for varying periods of years. In the case of osseous malunion no dentures that could offer the slightest masticatory value were possible owing to the greatly contracted and V-shaped nature of the

mandible. The encroachment upon the tongue space was met by a shrinkage of the tongue itself, thus obviating an additional misery for the sufferer.

In the cases of fibrous union, bridgework, where applicable, gives great comfort from the jaw rigidity and masticatory powers that it affords, but, of course, there are limits to its longevity. An example of fibrous union in which bridgework was impossible has been under my observation for upwards of fifteen years. An interest attaches to the case in that the patient always looks well nourished despite the fact that the mandibular portions are freely movable and remain separated from one another by a space of an inch and a half on one side. No apparatus has succeeded in establishing anything like rigidity in the jaw. The best that has happened has been a steadying power and help to the muscles in co-ordinating movements of the jaw. Of several dentures tried, the one that has answered best has been one of vulcanite with the block fitting into the space faced with soft rubber. This soft rubber has required renewal on three occasions during fifteen years. In cases where only a small loss of bone has occurred —e.g., an inch or under—especially if it is in the region of the symphysis, I should allow the bone ends to unite by osseous tissue, and afterwards endeavour to correct the appearance and "bite" by means of a denture. But the whole question of treatment turns upon the success or non-success of bone-grafting. Given a successful means of bone-grafting no one would allow mal-union of any kind to occur. And if by chance mal-union had occurred, an operation for refracture, or for refreshing the bone ends, would be undertaken preparatory to fixing a graft. I watch with great interest, therefore, the attempts at bone-grafting that are being made. I have no personal experiences to relate, but if a choice of bone from which to take a graft were forced upon me, I should elect for my tibia rather than for my rib. Preferably, however, I should like a graft taken from a young bone.

Time will not permit of my touching upon the vast subject of treatment of maxillo-facial deformities caused by this War. Plastic surgery in the hands of the skilful surgeon, assisted by mechanical art on the part of the dental surgeon—and we must not forget to add in this connexion the wig maker and glass-eye maker—will do much in the future for the relief and improvement of these distressing cases. I merely mention the subject in the hope that members will be able at future meetings to show patients exhibiting successful results.

In concluding these remarks, which I fear have been somewhat discursive, I am conscious that, to those of you who are daily engaged in war practice, they may appear to demonstrate the obvious. I must beg forgiveness, on the ground that my zeal, in relating personal experiences, has outrun what may be termed my presidential discretion. I feel that I am still a student, and have much to learn from the experience of others in war work; hence I look forward with pleasure to my period of office, as your President, as a fitting opportunity for an increase of knowledge.

A Report on the Inter-Allies Dental Congress, held in Paris, November, 1916, was presented and read by Mr. T. A. COYSH.

(*November* 27, 1916.)

Fuso-spirillary Peri-dental Gingivitis.

By Frank E. Taylor, M.D., and W. H. McKinstry, M.B., Captain R.A.M.C.

During the last few months large numbers of officers and men suffering from sore throat have been sent to the laboratory at the Queen Alexandra Military Hospital for bacteriological examination. As the result of the investigation of these cases, we have found spirilla and fusiform bacilli co-existing with or without a variety of other organisms, both cocci and bacilli.

The association of fusiform bacilli and spirilla with sore throat, often of a membranous or pseudo-membranous character, was first pointed out by Plaut in 1896, whilst the condition and its causal organisms were more carefully and completely described by Vincent in 1898. Since that date this inflammatory condition of the throat has generally been designated Vincent's angina, and the causative organisms have become known as the spirillum and *Bacillus fusiformis* of Vincent. The condition in Vincent's angina is usually an ulcerative or pseudo-membranous pharyngitis, and smear preparations taken from the affected parts, after proper fixation and staining, readily show the presence of the characteristic organisms of Vincent.

A further and more critical study of the cases sent to us revealed the fact that this condition was not always confined to the pharynx, but might also be present, as other observers have noted as well, on the mucous membrane of any part of the mouth and gums, constituting a fuso-spirillary stomatitis and gingivitis. In some of these cases the gingivitis is widely spread over the surface of the gums, but in others the ulcerative condition is confined, more or less closely, to the portion of the gums in close contact with the neck of the teeth, so as to constitute a real peri-dental or marginal gingivitis. It is to this condition of ulcerative fuso-spirillary peri-dental gingivitis and its relationship to Vincent's angina of the pharynx and to the character of the organisms found in the two conditions, that we desire to call attention in this communication.

An examination of the gums and teeth in patients the subjects of this infection usually reveals the following conditions: The teeth are frequently tartarated and unclean, showing evidence of great neglect—although this is not invariably the case, as in many of our patients the sound and well-groomed condition of the teeth was particularly noticeable. In an advanced and well-marked case the gums are red, swollen, and ulcerated in immediate contact with the neck of the teeth, and extending out for a variable distance, seldom exceeding ¼ in. The ulcerated surface is often covered with a soft, grey, foul-smelling exudation. The slightest touch with a swab or platinum loop to the ulcerated surface causes profuse bleeding. Any attempt on the patient's part to use a toothbrush also causes free bleeding, so that it must be temporarily discarded. The extent of the ulceration varies greatly, being limited in some cases to the neck of one or two teeth, in others extending the whole length of the upper or lower gums. The infection seems to show predilection for certain situations, being most frequently found about the lower central incisors, and behind the last lower molars. It is also frequently found about crowned and stopped teeth. In some cases the ulceration is limited to the tops of the papillæ between the teeth and to the parts of the gum separated from the teeth. In the slighter cases the ulceration around the necks of the teeth may not be readily visible, though the patient may only complain of bleeding of the gums on cleaning the teeth with a toothbrush. In these cases the affected areas are readily found and exposed by lightly rubbing the gums with a pledget of cotton wool, when oozing of blood immediately occurs. In the more severe cases, in addition to the bleeding, the patients complain of tenderness on pressure, and on mastication: whilst in two or three very long standing cases the affected teeth have become quite loose. In the majority of our cases the ulceration has developed slowly, and been of long standing, the patients stating that the gums have bled readily, and a foul taste has been present in the mouth for many weeks, months, or even years, and it is in these latter cases that the loosening of the teeth has been observed. In a few cases, on the other hand, the disease seems to run a more acute course, extensive ulceration developing in a few days. Hence the cases can be classified as acute or chronic. However developed, whether rapidly or slowly, the same condition results. The temperature in the uncomplicated disease is never raised, nor are the lymphatic glands enlarged or tender.

With regard to the ætiology of this affection, its wide-spread

prevalence amongst the troops in London at the present time suggests its infectivity, which is definitely proved by the results of bacteriological examination. In the majority of our cases the men were living in barracks—possibly overcrowded as the result of war conditions—but some came from camps, some from billets, and not a few were officers resident at their own homes. In some of these cases we found both husband and wife infected. A very similar, if not identical, condition clinically is extremely prevalent, we are given to understand, among both the British and French troops at the Front, being especially widespread in the French Army. The dental surgeons, we believe, find these cases very refractory to treatment, and are in the habit of speaking of them as "trench gingivitis," or "trench gum." We are not aware that this condition has been investigated bacteriologically, but from various descriptions we have heard, we are of opinion that the bacteriological findings would probably be the same as those we have obtained and describe here. That toothbrushes may become infected we have proved by finding the organism in the toothbrushes used by a few of our patients, and there can be little doubt that all articles coming in contact with the mouth may be similarly infected and become foci of infection.

Only the other day, and after this paper was written, the following suggestive history, indicating how the infection might be spread by toothbrushes, was brought to our notice. Sixteen men in billets in a certain private house were in the habit of keeping their toothbrushes on a common shelf. We have already had three of these sixteen men for treatment, and have examined the toothbrushes of two of them with positive results. About four weeks ago the third patient accidentally used one of the brushes since found to be infected, and to this he attributes his complaint, the gums becoming sore and bleeding about one week later.

Diet does not seem to play any part in the production of this disease, and it apparently has no relation to scorbutic affections of the gums, as fresh vegetables, or fresh fruit, or usually both, had been taken by all our patients.

The suggestion that lack of cleanliness is the important causative factor is entirely negatived by the number of cases we have seen in which the condition of the teeth themselves proves that hygienic measures had been systematically and scrupulously carried out.

The diagnosis of the disease is a comparatively simple matter. The character of the ulceration of the gum in close proximity to the neck of

the teeth, with special predilection for the lower central incisors and wisdom teeth, the overlying soft greyish exudation, the readiness with which bleeding is elicited, and the fœtor of the breath, all provide a clinical picture by which the disease can readily be recognized. This diagnosis is readily confirmed by a bacteriological examination, the microscope revealing the presence of fusiform bacilli and spirilla—both Gram-negative organisms, either alone or together with a variety of other organisms—cocci, bacilli, and leptothrices.

Many of our patients had already been under treatment, often for a considerable time, usually without much improvement, if any at all. They came to us with a diagnosis of pyorrhœa already made, and in some cases had been advised to have their teeth extracted. Now in this condition oozing of pus from the tooth sockets, and the presence of pockets of pus, as seen in pyorrhœa, do not occur, and we consider that this disease should be sharply differentiated from pyorrhœa. The clinical history, the treatment, and the prognosis of the two conditions are totally different.

As regards treatment, this is not commenced until the diagnosis has been established by bacteriological examination. Only those cases in which the typical Gram-negative fusiform bacilli and spirilla are found are included under this heading. In those cases in which the teeth are unclean and tartarated, the patient is sent to the dental department, where the teeth are scaled, and the tartar and membranous deposit are removed from the gums with a glycothymoline spray, under a pressure of 30 to 40 lb. to the square inch. The gums are then carefully dried with pledgets of absorbent wool, and afterwards painted freely with a 0·2 per cent. alkaline solution of salvarsan or kharsivan as prepared for intravenous injection. The drying and painting of the gums are then repeated at least once, and preferably twice, daily. On account of the tenderness of the gums, the scaling of the teeth may need to be done at two or three sittings, the swabbing of the gums with salvarsan being carried out between the sittings. Usually marked improvement is noticed in twenty-four to forty-eight hours, and the disease yields entirely to treatment in the majority of the cases within fourteen days if daily applications have been thoroughly and systematically carried out. In a few cases where improvement appears to be somewhat delayed under this treatment, we vary the medicament which is applied locally, the most successful results being obtained with a mixture containing vinum ipecacuanhæ, liquor arsenicalis, and glycerine. Other preparations also giving good results we have found to include: 10 per

cent. solution of quinine and colloidal preparations of iodine and silver (collosol iodum or collosol argentum). In a few cases, chiefly in officers remaining on duty during treatment, more prolonged attention has been found necessary, but in these cases the attendance of the patients was irregular, and so prevented that regular and systematic swabbing which we have found essential for success. In all cases mouth-washes of peroxide of hydrogen, or chlorate of potash, with carbolic acid, were prescribed to be used freely after each meal. Patients were also advised to destroy their toothbrushes, pipe mouth-pieces, cigarette-holders, and all articles coming in contact with the mouth, and obtain new ones at the end of their course of treatment. In one or two cases the dental surgeon has found it necessary to remove carious teeth, and in one of these where the gums were badly ulcerated—a deeply excavated ulcer also existing behind the last lower molar which was extracted—we were able to demonstrate the presence of a fusiform bacillus at the apex of the root of the tooth.

During the course of treatment outlined above, marked, and as a rule, rapid improvement is observed—the ulceration of the gums heals, the pain, the bleeding, and the fœtor of the breath all disappear, and where the teeth have been loose, they tighten up again. In no case have we found it necessary to have the teeth extracted for this condition. Microscopic observations show that along with this improvement there goes *pari passu* a diminution of the number of fusiform bacilli and spirilla, and when the cure is complete, these organisms have entirely disappeared. No case is discharged by us until it has been proved microscopically to be free from both the spirillar and the fusiform organisms.

To various members of the staff of the Queen Alexandra Military Hospital, and especially to our dental colleagues, Sir Francis Farmer and Captain Johnson, R.A.M.C., we are indebted for many of our cases and for the interest they have displayed in the results of our work.

DISCUSSION.

Mr. PERCY EDGELOW: I am much interested in Dr. Taylor's and Captain McKinstry's paper, having attended a considerable number of officers of the Royal Flying Corps for acute septic gingivitis. I had entertained an impression that the particular nature of their avocation was in some way responsible for the prevalence of the trouble, but after the authors' remarks as to the frequency with which he has come across it in other branches of the Army, it is obvious that the particular conditions surrounding "flying" will not suffice to explain these attacks. I agree with him that this gingivitis is an affection distinct from pyorrhœa alveolaris, though no doubt they are to be found co-existent in the same mouth. I also have found that the gums and soft tissues around the wisdom teeth are very liable to be affected, particularly in the lower jaw, with accompanying swelling and tenderness of the neighbouring pharyngeal mucous membrane, a painful submaxillary gland being often present. The adjacent mucous membrane of the cheek is occasionally so swollen as to intrude between the opposing molars and is lacerated when biting, causing a very painful inflamed and ulcerated condition and leading to difficulty in opening the mouth beyond one third of its normal width. The fœtor of the breath is great and there is often pronounced general malaise. The swollen condition of the gums leads to free bleeding on the slightest instrumentation, and to the formation of pockets between the necks of the teeth, particularly in the molar and premolar regions. Marginal sloughs are frequently present. Solid food is avoided in very acute cases on account of the pain during biting, and fluid nourishment alone is tolerated. The affection is apparently uninfluenced by the actual condition of the teeth as regards caries, many of the acutest attacks coming under my notice having occurred in mouths possessing a full and sound set of teeth. As regards treatment, I first give an iodine wash to rinse the mouth thoroughly and then freely apply a mixture of equal parts of carbolic acid and camphor. The camphor prevents the caustic and irritating effect of carbolic acid used in a strength of 1 in 2. The extreme tenderness and free bleeding quickly yield to this treatment. For home use I rely on mouth-washes of carbolic acid and alum, or permanganate of potash. In exceptionally severe cases with much swelling and ulceration of the mucous membrane near the wisdom teeth accompanied by laceration of the cheek mucous membranes, I have extracted the upper wisdom tooth under novocaine with rapidly beneficial results. I select the upper wisdom tooth for three reasons: (1) It articulates with the lower wisdom tooth alone, whilst the latter has the benefit of articulating with both the second and third upper molars; (2) it is much easier to extract than the lower wisdom; (3) owing to its better drainage the upper wound heals more rapidly than the lower one.

Mr. SCHELLING: Are cultivation and other means requiring considerable time, necessary to confirm the existence of the micro-organisms described? For the cases which I have been called upon to treat presenting a similar appearance to those just described are generally those of officers, home on six or ten days' leave, who are anxious to have as much relief as possible in the fewest number of visits. The treatment adopted consists of, at first visit: (1) Free swabbing with peroxide of hydrogen 10 vols.; (2) complete painting with lin. iodi. applied on a small pellet of cotton and used both for its antiseptic action and its property of staining all the tartar so that it can be more easily removed with appropriate fine scalers; (3) polishing with a buff and pumice powder; (4) seeking for pockets and packing them with cotton plugs moistened in saturated solution of chinosol, the pockets behind the wisdom teeth being similarly packed with small plugs moistened in pure carbolic acid. A second visit next day to remove the plugs and complete the scaling and apply iodine, and if necessary more visits for chinosol dressings in pockets on each occasion and, finally, instructions to the patient thoroughly to brush the gums inside and out daily with a palate brush and a little table salt and to return for inspection at the next opportunity.

Mr. A. PARKER CATER (Lieutenant G. B. C., 36 Corps d'Armée française): One of many experiences in connexion with the work and organization of the Dental Operating Vans which I took to France, for service with the French Army at the Front, has been the large and increasing number of cases of what we called "trench gingivitis" and known on "this side" as "trench gum." Whatever may be its correct nomenclature the fact remains that its lowering effects on the men's general physical condition and efficiency is most marked. Not only does it attack the gums around the teeth but it also shows itself in patches of varying size over the gums and inside the lips and cheeks according to the severity of the case. At a certain stage it might have been mistaken for "pyorrhœa" pure and simple, but the extreme rapidity with which the symptoms developed appeared to me to suggest a specific organism as being the actual cause of the complaint. The "services" of these motor dental surgeries being chiefly devoted to men *coming out of* trenches led one at first to think that the trouble might possibly be confined to the front line troops. I am now, however, constantly discovering that the complaint is spreading not only to men who have never been near the trenches or even out of England, but also to civilians. In practically all these cases it appears to be pretty certain that they have been in actual or indirect contact with infected individuals. It has been suggested to me that it is invariably caused by neglect or want of cleanliness. As against this, and also to demonstrate how highly infectious it appears to be, I have frequently found it in mouths where there was no lack of cleanliness. As I have had attacks myself, one may fairly anticipate that this would not be brought about by any neglect on my own part. As already stated, I am of opinion that some special organism is the active cause of the trouble. It is highly infectious and

need not necessarily be caused by lack of, or inability to secure, "oral" cleanliness, because if such lack of cleanliness was the dominating cause of the trouble one might expect, practically, nearly every man at the Front to be affected. The treatment indicated as far as my experience goes appears to be thorough scaling and cleaning as soon as the tenderness permits; frequent irrigation under pressure; applications of liquor arsenicalis with vini ipecac. and glycerine alternated with salvarsan, also iodine; frequent and *proper* use of *clean* toothbrushes and vibratory or digital massage. In stubborn cases I found that ionic medication yielded quick and satisfactory results. Educational treatment consists in teaching men to avoid using each others drinking bottles or other utensils, which in spite of Army Regulations they persist in doing; the passing on of pipes, cigarettes, towels, &c. The renewal of toothbrushes should also be more frequent. As far as possible a rapid examination of troops leaving trenches for rest camps has been made, and on several occasions, I have found that a man with a comparatively clean mouth, would, in two or three days, develop an acute condition of trench gum, with putrescent discharge around necks of teeth, bleeding and swollen gums, teeth loose and so tender as to be useless for masticating; fœtid breath, furred tongue and general feeling of "malaise" and slackness. If a man develops this acute condition in the trenches, which he frequently does, he becomes of little practical use as a fighting unit, he cannot tackle his trench rations, loses nerve and also becomes an infectious menace to his fellows. The "toilette" of the mouth whilst in the trenches is very difficult, and often impossible, unless men can be provided, as part of their kit, with a paste in the form of kolynos, or some such preparation, —which they are not. As a great many civilian, as well as military, dental surgeons are now constantly having to deal with this complaint, it will be very helpful and interesting to obtain their experiences and opinions on the subject. At one base consisting mainly of chefs, and cooks engaged in bread-making, we recorded no cases of trench gingivitis. There were nearly 700 of these cooks. I have not found any cases of the complaint in men suffering from *fractures* or *traumatic* injuries of the mouth, either in France or on this side.

Dr. J. D. ROLLESTON: Is not the condition described by Dr. Taylor and Captain McKinstry the same as that described by Vincent as "ulcero-membranous stomatitis," which is due to the same organisms as Vincent's angina, and was occasionally associated with it?[1] Being attached to the Val de Grâce military hospital, Vincent especially noted its occurrence amongst soldiers and its liability to arise in connexion with the last molar tooth. It was frequently seen among the recruits from Normandy and Savoy whose teeth were predisposed to caries by drinking cider. As medical officer in a fever hospital I have been interested in the condition for many years.[2] Like Vincent's angina, it may occur as a primary condition or be secondary to

[1] Vincent, *Compt. rend. Soc. de Biol.*, 1904, lvi, p. 311.

[2] J. D. Rolleston, *Brit. Journ. Child. Dis.*, 1910, vii, p. 299.

some other acute infection such as scarlet fever or measles. I agree with what the authors have said as to the absence or slight degree of general disturbance. I lay special stress on the peculiar fœtor as of diagnostic value. As regards what has been said about the occurrence of fusiform bacilli in suppurative conditions, it may be noted that Vincent found them in a number of suppurative processes in connexion with the alimentary tract as well as in a case of pyothorax.[1] What relation do the authors think this condition bears to cancrum oris, which I regard as a further stage of ulcero-membranous stomatitis, just as gangrenous angina is a further stage of Vincent's angina?[2] In all these conditions the fuso-spirillary symbiosis is met with. As regards prognosis and treatment I have found that the condition as a rule is mild and yields readily to simple treatment, such as applications of tincture of iodine, methylene blue, or a mouth-wash of potassium chlorate and myrrh. In rare cases local application of salvarsan[3] may be required.

At the conclusion of the discussion, the authors accepted the President's suggestion that the word "gingivitis" should be used instead of "periodontitis."

[1] Vincent, *Comp. rend. Soc. de Biol.*, 1905, lvii, p. 772.

[2] J. D. Rolleston, *Brit. Journ. Child. Dis.*, 1912, ix, p. 314.

[3] J. D. Rolleston, *Proceedings*, 1913, vii, Clin. Sect., p. 1; and *Practitioner*, 1913, xci, p. 847.

Section of Odontology.

President—Mr. W. B. Paterson, F.R.C.S., L.D.S.Eng.

(*January* 22, 1917.)

Case of Facial Restoration by Means of a Mechanical Appliance.

By J. Lewin Payne, M.R.C.S., L.D.S.

The patient was a corporal in the West Yorkshire Regiment, aged 24, who received a gunshot wound on November 17, 1914, which carried away the whole of his upper lip, a piece of the nose and the anterior portion of the superior maxilla; the mandible was fractured on the left side in the first molar region. The upper lip, with the portion of the maxilla and the teeth attached, was found in the trench the next day. During the first five months after the injury several teeth and pieces of bone were removed from the mouth, but he received no special treatment from a dental surgeon.

In April, 1915, the patient was sent to Guy's Hospital, under the care of Sir Arbuthnot Lane. Owing to the large amount of hard and soft tissue that had been lost, and also in consequence of the long interval that had elapsed since the injury was inflicted, Sir Arbuthnot Lane was of opinion that a plastic operation was not advisable, and he asked me to see the patient with the object of fitting an appliance.

Suppuration was still going on when I first saw him and several small sequestra and septic roots were found in the mouth. In the superior maxilla everything in front of the second premolar on the right side and in front of the second molar on the left had been lost. The patient was very depressed, he avoided seeing other people in the ward, and insisted upon wearing a muslin mask below the eyes so as to cover his deformity. The septic roots and a few small sequestra were removed under an anæsthetic without delay and the mouth was kept constantly irrigated. Suppuration soon stopped. A temporary appliance was fitted two or three weeks after the extraction of the roots. This consisted of an artificial vulcanite lip attached to a

vulcanite denture which was held up by means of clasps. To the artificial lip a moustache was attached. A denture was also fitted to the lower jaw.

In November last the patient came to the Maxillo-facial Hospital at Kennington to have his permanent appliance fitted. He now wears that appliance, which is similar to the temporary appliance except that the lip portion is detachable and is made of aluminium with two parallel rods fitting into tubes in the front of the upper denture.

Mr. Howard Mummery kindly undertook the colouring of the portion of the artificial lip which remained uncovered by the moustache.[1]

(*January* 22, 1917.)

Mandibular Anæsthesia.

By A. E. Rowlett, L.D.S.Eng.

Local anæsthesia, and more especially conductive local anæsthesia, has not received in this country the attention which, I venture to say, it merits. For our information, drugs, instruments and technique, we are very largely indebted to foreign scientists, such as Braun and Fischer, and it is to their writings that I owe my introduction to the subject which is to occupy our attention to-night. The radiographs showing the needle in position I had thought an original idea, and it was a disappointment to find that such photographic representation had already been carried out by Thoma.

The advance in the technique of local anæsthesia for conservative operations in the mouth, in my opinion, marks an era in dentistry, and its intelligent application will help to solve many of the difficult problems that confront our profession. Local anæsthesia of both maxilla and mandible would present too large a field for anything like thorough treatment in the limited time at our disposal. Mandibular anæsthesia appealed to me because, before I adopted the technique to be described, dentinal anæsthesia in the lower jaw, and especially of the lower molars, was generally a matter of considerable difficulty. My aim is to present my subject before you in such a way that you may be able clearly to visualize an operation which can only be perfectly explained

[1] Two illustrations of this case appeared in the *Proceedings*, 1916, ix (Sect. Odont.), p. 76, and which are reproduced (figs. 1, 2) together with another showing present condition of the patient (fig. 3), on p. 40 of this number.

by the aid of clinical demonstration. To this end we will first consider the anatomy of the mandible, paying particular attention to these parts of the osseous framework, and of the soft tissues which are intimately connected with the technique of injection and the results obtained from such injection.

The Bony Mandible.

The internal surface of the body of this bone is smooth and dense around the roots of the teeth, and the only canaliculi to be found are those beneath the incisor teeth. These canaliculi communicate with the mandibular canal and the absence of such foramina is a contra-indication for infiltration anæsthesia. About the middle of the ascending ramus is seen an irregular shaped opening—the inferior dental foramen, which transmits the inferior dental vessels and nerve. This canal runs downwards and forwards into the bone, and its anterior margin is prolonged into a sharp ridge to which is attached the internal lateral ligament of the jaw. The course of the canal in the bone is thus described by Gray: "The inferior dental canal runs obliquely downwards and forwards in the substance of the ramus, and then horizontally forwards in the body; it is here placed under the alveoli with which it communicates by small openings. On arriving at the incisor teeth, it turns back to communicate with the mental foramen, giving off two small canals which run forward, to be lost in the cancellous tissue of the bone beneath the incisor teeth. This canal in the posterior two-thirds of the bone is situated nearer the internal surface of the jaw; and in the anterior one-third nearer its external surface. Its walls are composed of compact tissue at either extremity and of cancellous tissue in the centre." The upper part of the canal is expanded into a fairly well-defined fossa, which is, of course, superior and posterior to the opening of the canal, and this is known as the pterygo-mandibular fossa, from its relation to the mandible and the internal pterygoid muscle.

The following muscles claim our attention: The internal pterygoid, attached at the angle running upwards and inwards to its origin in the pterygoid fossa, the palate bone and its tuberosity, and tuberosity of the superior maxilla. It is convenient to consider this muscle internally as corresponding to the masseter muscle externally, with the ascending ramus of the mandible between. The space between the internal pterygoid muscle and the mandible, which contains the inferior dental vessels and nerve embedded in loose connective tissue, is most important in conductive anæsthesia. The mylohyoid muscle, attached

to the mylohyoid ridge, is of interest, because of its nerve supply, a small branch of the inferior dental. A few fibres of the superior constrictor are attached to the alveolar border above the mylohyoid ridge. The temporal muscle is attached to the coronoid process, and these muscles may be pierced by the needle whilst injecting for anæsthesia of the inferior dental nerve. The external pterygoid muscle is attached to the pterygoid fossa of the condyloid process. The external surface of the mandible, according to Fischer, always exhibits sieve-like perforations on the alveolar ridges at the necks of the teeth; the cancellous areas are limited to the anterior portion in the mental region. As one would expect, these foramina tend to disappear, and the bone to become denser with age. Midway between the alveolar border and the lower border of the mandible, and immediately between the two bicuspid teeth, is placed the mental foramen, which transmits the mental vessels and nerve. This canal runs inwards, downwards and forwards, and communicates with the inferior dental canal. The buccinator muscle is attached to the alveolar border opposite the three molar teeth, and the masseter to the lower half of the ramus and angle of the jaw. The external oblique line runs upwards and backwards from the base of the mental process to be continuous with the anterior border of the ramus. The anterior border of the coronoid process presents two ridges, the inner one of which is merged into the internal surface of the process. The triangular space between these two ridges, and posterior to the last molar tooth, is known as the retro-molar fossa.

When looking at the mandible anteriorly, we should particularly note that the internal oblique line is situated externally to the line of occlusion, and also, that the anterior portion of the internal surface of the ramus is inclined inwards and backwards, and does not face directly inwards.

The Inferior Maxillary Nerve.

The inferior maxillary division of the fifth cranial nerve arises from the Gasserian ganglion, and enters the pterygo-maxillary region through the foramen ovale; it is mixed nerve trunk, the motor root joining the sensory nerve just external to the foramen ovale. It lies here under the external pterygoid muscle, and after a short course divides into an anterior and posterior branch. Before division it gives off two branches, the nervus spinosus and the nerve to the internal pterygoid. The smaller anterior division is almost entirely motor, and supplies the muscles of mastication; its branches are masseteric, deep temporal, long buccal

and internal pterygoid. The posterior division is almost entirely sensory, and has three branches; the auriculo-temporal, generally arising by two roots which embrace the middle meningeal artery; the lingual nerve and the inferior dental. With three of these branches we are very particularly concerned—the buccal, the lingual, and inferior dental; they, therefore, demand a somewhat detailed description.

The buccal, or long buccal, runs between the two heads of the external pterygoid muscle, to which it supplies a branch, beneath the anterior border of the masseter, to reach the outer surface of the buccinator muscle, the integument and the mucous membrane lining the internal surface of the muscle.

The lingual nerve lies at first beneath the external pterygoid muscle together with the inferior dental nerve, being placed to the inner side of the latter nerve; it then passes between the internal pterygoid muscle and the inner surface of the ramus and crosses obliquely to the side of the tongue running to its apex, lying immediately beneath the mucous membrane. It supplies the mucous membrane of the mouth, the gums, the sublingual gland, the fungiform and filiform papillæ and mucous membrane of the tongue.

The inferior dental nerve, the largest of the branches of the inferior maxillary nerve, comes from under the external pterygoid muscle, lies on the internal lateral ligament of the lower jaw, and enters the inferior dental canal. It is accompanied by the inferior dental artery and veins.

Its branches are: (*a*) Mylohyoid, given off just before entering the foramen; (*b*) dental, supplying the molar and bicuspid teeth; (*c*) incisive, which is continued onwards in the bone to the middle line and supplies the canine and incisor teeth; (*d*) mental, which emerges from the mental foramen, and supplies the skin of the chin and the skin and mucous membrane of the lip. This nerve at the middle line communicates with branches of the nerve of the opposite side.

Instruments.

For conductive anæsthesia, where we are often injecting somewhat deeply among the tissues and approaching important vessels and structures, it is very necessary that as perfect asepsis as possible be maintained both in the solution and in the instrument used for injecting the solution.

The syringe should be constructed entirely of metal or of glass and metal. Most of the dental syringes upon the market are unsuitable,

since they contain washers of either leather, rubber or some other packing. These washers are very difficult to keep clean, do not stand boiling well and deteriorate when kept in spirit. It has also been the custom to lubricate such syringes, and I think that it is evident that such syringes leave much to be desired from an aseptic point of view. For some of the injections used in dental work considerable pressure is necessary, and the ordinary aseptic syringes upon the market are neither made with sufficient accuracy of fit, nor are they constructed so as to permit the fluid to be expelled with sufficient force. These objections do not apply so strongly in mandibular anæsthesia as no great pressure is needed for this injection.

Undoubtedly the best dental syringe yet constructed is the Fischer syringe, but of course this is not obtainable at the present time. Mandibular anæsthesia, however, can be perfectly well performed by the ordinary surgical aseptic syringe, consisting of a glass barrel with metal mounts and a metal plunger. The usual capacity of the syringe is 2 c.c., and it is fitted with a push-on attachment, the needle sliding on to the syringe and not screwed on as is the case of the ordinary dental syringe. An adapter is made by which the ordinary aseptic needle can be used if it is desired, or an irido-platinum needle with the usual push-on mount is quite satisfactory. Probably an all-metal syringe is most satisfactory, and this is the opinion of Braun. Glass barrels will break at times even with the greatest care, and certainly all glass and metal syringes should be constructed so that a broken barrel can be replaced by the operator in a few minutes. It is wise to have two or three syringes in readiness, as much time is saved in this way.

It is desirable to have long and short needles, the long needle 45 mm., and the short one the length of the ordinary aseptic needle, about half that length. In my hands the irido-platinum needles have proved much more satisfactory than steel; I do not find irido-platinum needles too soft, although of course they need careful handling, and different makes certainly vary in rigidity. This is probably due to the varying proportions of the alloy.

Syringes and needles may be kept sterilized and ready for use in spirit; it is very convenient to have one syringe fitted with a short, and one with a long needle to avoid waste of time.

In addition to syringes and needles, I use small porcelain gallipots, which can be boiled and which are used as containers; graduated test tubes, for dissolving the solution; a flask containing Ringer's solution,

and a flask containing sterile water for washing spirit out of the syringe and needle; tubes containing the drug in tablet form, and a pair of sterile conveying forceps.

The Injecting Solution.

The drug I have used exclusively for mandibular anæsthesia is novocain combined with adrenalin or synthetic suprarenin. Owing to the difficulty of obtaining tablets until recently, I was compelled to have a solution made from novocain powder, of which fortunately, I possessed a considerable stock; to this solution I added adrenalin. Although my solutions were fairly fresh, the results were not so satisfactory as those I obtain from tablet solutions. I use a 2 per cent. solution in all cases, and generally suprarenin 1 in 20,000. Braun says that not more than 40 c.c. of a 2 per cent. solution should be injected—that is, 8 grm. For suprarenin the dose is given as 5 mg. At the Royal Infirmary, Leicester, where a good many major operations are performed under local anæsthesia, 20 c.c. of a 2 c.c. solution combined with adrenalin, of a considerably greater strength than 1 in 20,000, has been injected, and no toxic symptoms whatever were noticed. These are quantities far in excess of those which the dentist will require to use.

The toxicity of novocain in the doses used in ordinary dental practice is negligible; the question of novocain combined with suprarenin is somewhat different. Certain individuals show an extraordinary susceptibility to this drug, and in such cases it is wise to reduce the dosage. Thoma is also of this opinion, and strongly supports the view as to the advisability of keeping the proportion of suprarenin low. Fortunately it is easy to pick out cases of susceptibility as the symptoms show themselves almost immediately, and will subside if the syringe is quickly withdrawn.

Another point to be remembered is that the same patient may react quite differently to a similar dose on different occasions; this is not to be wondered at, when one considers how differently tobacco will affect the same person at different times. Further, I have remarked that the effects of the drug are nothing like so marked when the fluid is deposited in a space filled with areolar tissue as when the injection is made into the tense fibrous tissues of the gums or palate. There is even a greater reaction noticeable with Parrott's method of injection, especially when used in the lower jaw. I feel quite sure that all the toxic symptoms I have observed are referable to the hæmostatic agent and not to the anæsthetic drug.

The solution must of course be isotonic with the blood or nearly so, and it is found that the best results are obtained by dissolving tablets in 7 per cent. salt solution or in the modified Ringer's solution: Sodium chloride, 0·5 grm.; calcium chloride, 0·04 grm.; potassium chloride, 0·02 grm.; aqua destillata, 100 c.c. The tablets are of two kinds, E and F: E tablet contains novocain, 0·02 grm.; suprarenin synthetic, 0·05 grm. F tablet contains novocain, 0·05 grm. Tablet E when dissolved in 1 c.c. of normal saline, or Ringer's solution, makes a 2 per cent. isotonic solution of novocain and with $\frac{1}{20000}$ synthetic suprarenin. Tablet F when dissolved in 2·5 c.c. of normal saline or Ringer's solution makes a 2 per cent. isotonic solution of novocain. If it is desired to reduce the dose of suprarenin, it is only necessary to add a portion of the F solution to the E solution. Thus if 1 c.c. of F solution be added to 1 c.c. of E solution a 2 per cent. solution containing suprarenin $\frac{1}{40000}$ is obtained and by increasing the proportion of the E solution the suprarenin may be diluted to any extent. There is a growing tendency to reduce the proportion of suprarenin in solution and the strength of 1 in 20,000 is only recommended when profound anæmia is required.

The technique which is used for preparing a solution is as follows: Into a sterile test tube is poured 1 c.c. of Ringer's solution for each E tablet, or 2·5 c.c. of Ringer's solution for each F tablet, to be dissolved. The solution is heated to boiling point, and the tablet or tablets dropped in with sterile forceps. If less than $\frac{1}{20000}$ suprarenin is required, the E solution is diluted with the F solution to the required extent. The solution is then poured into the sterile container and taken up into the syringe, which has previously been thoroughly washed through with distilled water.

Technique of Injection.

The operator must be able exactly to visualize the anatomical structures into which he is about to plunge his needle. He must especially bear in mind that the point at which the needle should pierce the mucous membrane is on the internal oblique ridge and about 1 cm. above the occlusal surface of the last molar, and also that the internal surface of the ascending ramus of the mandible faces backwards as well as inwards. In some jaws this deflection is more marked than in others.

I will first describe the technique for injecting on the right side. The operator stands on the right and in front of the patient. With

the forefinger of the left hand palpate the internal oblique line; now, still pressing, move the finger slightly externally, so that the mucous membrane is made tense over the ridge, and space is left for the needle to enter. Swab the field of injection with tincture of iodine to sterilize it. Hold the syringe in the right hand with a pen grasp, and plunge the needle sharply into the mucous membrane. It is important that the needle should be sharp, and that a quick stab should be made so that the puncture is as painless as possible; this puncture of the mucous membrane is often almost painless, and is usually not nearly so painful as in the case of injections nearer the gingival margins. The point of the needle should now be touching the bone at the internal oblique ridge; without moving the syringe gently release the tension exerted by the forefinger until the point of the needle is sufficiently internal to be free of the bone; now swing the barrel of the syringe to the right, and advance the needle. If it is desired to anæsthetize the lingual nerve, start injecting at once, but the great bulk of the solution, say 1·5 c.c., should be injected into the pterygo-mandibular fossa. Whilst advancing the needle be sure to keep in contact with the bone; when the correct point has been reached, about 2 cm. from the internal oblique line, the needle will come to a full stop and it will be found difficult to advance further; inject the rest of the solution slowly, moving the needle gently backwards and forwards so as to distribute the fluid and avoid injecting into vessels.

Difficulties.—In some mouths the palpation of the internal oblique line presents certain difficulties. This line can always be found by running the forefinger along the sulcus on the outer surface of the mandible until the external oblique line is felt; now advance the finger round the last molar tooth, and posterior to this tooth a triangular depression, the so-called retro-molar triangle, will be felt; the inner border of this triangle is the internal oblique line. The danger of perforating arteries or veins is *nil* if the technique is correctly followed, and in any case the puncture of an arterial or even venous wall is a matter of considerable difficulty at any time, and when it is lying in loose connective tissue, almost an impossibility. The great danger is that of injecting too low or too near the middle line; injecting too near the middle line can easily be avoided with care, as the landmarks are so clearly defined. The former is more easily done, and in certain jaws it is by no means easy to calculate the exact point of insertion, and the fact that the lower incisors may be well above the occlusal plane must be taken into account. It will be noticed in such cases that the needle does not

enter so freely and that there is not the same definite point of contact with the bone as when the correct place of injection is reached.

Technique of Injection on the Left Side.—Stand behind the patient with the left arm around the patient's neck, and palpate the internal oblique line. Puncture the mucous membrane as before, and the procedure is the same as on the right side except that the syringe is swung to the right instead of the left.

Results of Injection.

If the injection has been correctly performed, the first symptom that the patient will notice is a feeling of numbness in the tongue and in the lower lip on the same side as the injection has taken place. If the whole of the fluid has been reserved for the inferior dental nerve, numbness of the tongue will probably be absent. A feeling of numbness in the lower lip is a sign that the injection has been successfully performed, and if it is absent successful anæthesia of the teeth cannot be hoped for.

The time required for full anæsthesia of the nerve varies considerably in different cases; usually the numbness in the lip is felt after about five minutes, and anæsthesia of the nerve is at its height in from fifteen minutes to half an hour. The teeth are anæsthetized in rotation from behind forwards, that is to say, molars first and incisors last; the duration of the anæsthesia is about forty-five minutes, and if operating upon several teeth it is wise to begin with the one most anterior, as the anæsthesia fades away in the reverse order to its onset.

If operating on incisors, an injection of 0·5 c.c. in the mental fossa upon the opposite side is often necessary owing to the communicating branches which cross the median line. For extractions it is also necessary to inject the long buccal nerve; 0·5 c.c. injected horizontally in the sulcus of the molar region is sufficient.

After thirty minutes has elapsed it is possible to cut simple cavities in any tooth in the lower jaw without pain; by simple cavities I mean cavities in which the nerve is not involved. Absolute anæsthesia of the nerve is present in many cases but not in all, but in practically all cases of successful injection the nerve can be freely exposed with no pain worth mentioning and then pressure anæsthesia applied.

If the periodontal membrane of a tooth is inflamed, the pain of the operation may be lessened, but it is not right to expect that it will be abolished. This applies to extraction as well as cavity cutting. If care

be taken in cutting cavities there is no danger of exposing nerves, and so far from causing pulpitis I believe that there is less danger of pain in teeth filled under local anæsthesia, since the stimulation of the pulp and its resultant hyperæmia, which is present in operating on sensitive teeth without any anæsthetic, is in these cases entirely absent.

If the periodontal membrane and pulp be healthy, I have obtained uniformly good results by immediate filling; of course in the case of inlays this is impossible, as it is also in the case of two approximal cavities.

In my hands the best temporary filling has been proved to be oxy-sulphate of zinc; in very shallow cavities a little paraform may be mixed with it; temporary gutta-percha seems to leave the dentine hypersensitive. It is wise to line all permanent fillings with oxyphosphate, as the absence of sensitiveness may mask a nearly exposed pulp.

Injection at the Mental Foramen.

For the injection at the mental foramen again use the long needle. On the right side the operator stands behind the patient. With the forefinger of the left hand palpate the mental foramen on the cheek, and with the thumb evert the lower lip so as to expose the gum beneath the two bicuspids. Sterilize the mucous membrane as before, and at a point in the sulcus between the two teeth, insert the needle and carefully advance until the mental foramen is reached. This will be indicated by slight pain in the area of distribution—i.e., the lower lip. Inject a few drops immediately, and exert pressure with the forefinger so as to force the solution into the foramen. Now swing the syringe back as far as possible, so that the needle may be inclined downwards, forwards and inwards; advance the needle carefully into the foramen, injecting at the same time, and inject in all about 1·5 c.c. Withdraw the needle and massage the gum so as to squeeze the solution as much as possible into the foramen.

On the left side stand in front of the patient and proceed as on the right side; but in this case palpate the mental foramen with the thumb and evert the lip with the forefinger.

If the injection has been successful, anæsthesia of the canine, incisors, first bicuspid and sometimes second bicuspid should take effect almost immediately, so as to allow free cutting of the dentine and extirpation of the pulp. For extractions a lingual injection is necessary as well.

After-pain from the Injection.

If the mandibular injection has been correctly and aseptically performed, there should be no after-pain. The most common cause of after-pain is infiltration of the muscle fibres of the internal pterygoid by injecting too low down too high a percentage of suprarenin. Other causes may be (1) stale solution, due either to drugs which have become deteriorated before the solution was made up or to chemical changes which have taken place since the solution was made; (2) lack of asepsis in solution, needle or syringe; or (3) septic condition of mucous membrane in which the puncture is made. This however is not a serious difficulty in the general application of local anæsthesia. It occurs but seldom, and if care be taken is never serious. The real difficulty, and the main objection raised by the patient, consists in the toxic symptoms which arise during the injection. These symptoms are due to deteriorated drugs, too high a percentage of suprarenin, and in some few cases the idiosyncrasy of the patient. In the hands of a thoroughly proficient anæsthetist these toxic symptoms should never be allowed to assume such proportion as to distress the patient. As soon as the first toxic symptoms become manifested by pallor, quickened pulse and respiration, the syringe should be withdrawn; if, after the suprarenin is reduced to a minimum dose $\frac{1}{100000}$ the symptoms still persist, the case should be deemed unsuitable for local anæsthesia, but such cases are very few and far between. Symptoms that are truly toxic must be carefully differentiated from purely mental effects, which closely resemble them.

Mr. Loughnane, house surgeon at the Leicester Royal Infirmary, who has a wide experience in the use of novocain and adrenalin for general surgery, claims that he has never experienced any severe toxic symptoms in their use. He has injected as much as ten syringefuls of 2 per cent. novocain, each syringe containing 2 c.c. of the solution, that is 0·4 grm., and as much as 2 c.c. of $\frac{1}{1000}$ solution of adrenalin without any marked toxic effects. It is true that in all cases the patient was in a recumbent position, but we may safely conclude that the dentist need have no fear of the toxic effects of the comparatively small amount of the drugs he may inject. Personally I have never injected more than 5 c.c. of a 2 per cent. solution combined with adrenalin $\frac{1}{20000}$.

A colonial dentist, whom I know from personal experience to be a keen and conscientious observer in all dental matters, tells me that after considerable experience in the use of local anæsthetics both in Queensland, Australia, and in the North of Scotland, he considers the

first symptoms which occur at the time of injection to be almost entirely mental. These symptoms he found to be much more marked in Australians, the inhabitants of a hot climate, than in the natives of the North of Scotland. He also experienced considerable help from the use of four or five drops of validol given in a wineglassful of water immediately before the operation.

With all deference to these observers, I am still of the opinion that the symptoms that occur on injection in the majority of cases are due to the action of the drugs used, and only in a very small minority of cases are they to be considered as purely mental. By diminishing the dose of suprarenin, I have found these symptoms much less frequently present; I have also observed that fresh solutions made from tablets and used immediately are less toxic than stock solutions which are of necessity comparatively stale.

Practical Application of the Method.

By far the most important application of mandibular anæsthesia, to my mind, is its use in the preparation of teeth for conservative work. For extractions we have a choice of anæsthetics—ether, nitrous oxide and oxygen, nitrous oxide alone and the local anæsthetics; the elimination of pain for the extraction of teeth is not one that seriously hampers the dentist in his daily work; indeed it may be said to have been solved.

Pain in conservative dentistry, if not the most difficult problem, is still one of the most difficult problems which confront the conscientious dentist in his daily work. I have had experience with the local application of ethyl chloride, the ether spray, various obtundents of which the most successful is paraform, the high-pressure syringe, Parrott's method of local anæsthesia, nitrous oxide, and oxygen analgesia; but none of these agents in my hands have proved anything like so efficient as conductive anæsthesia in the mandible. The more general application of some form of anæsthesia in conservative dentistry seems a natural step in the evolution of our specialty; how many teeth are sacrificed, or at best imperfectly filled, in deference to the humanity of the operator in dealing with sensitive tissues!

General surgery without anæsthetics is unthinkable, and the intelligent and skilful application of local anæsthesia will help to perfect the art of dentistry and stimulate that research which will one day make dentistry more truly a science.

I must not conclude without saying how much I owe in my

preparation of this paper to the house surgeon and residents of the Leicester Royal Infirmary, to the curator of the Leicester Museum, who found me the specimens, and to the co-operation of the photographer.

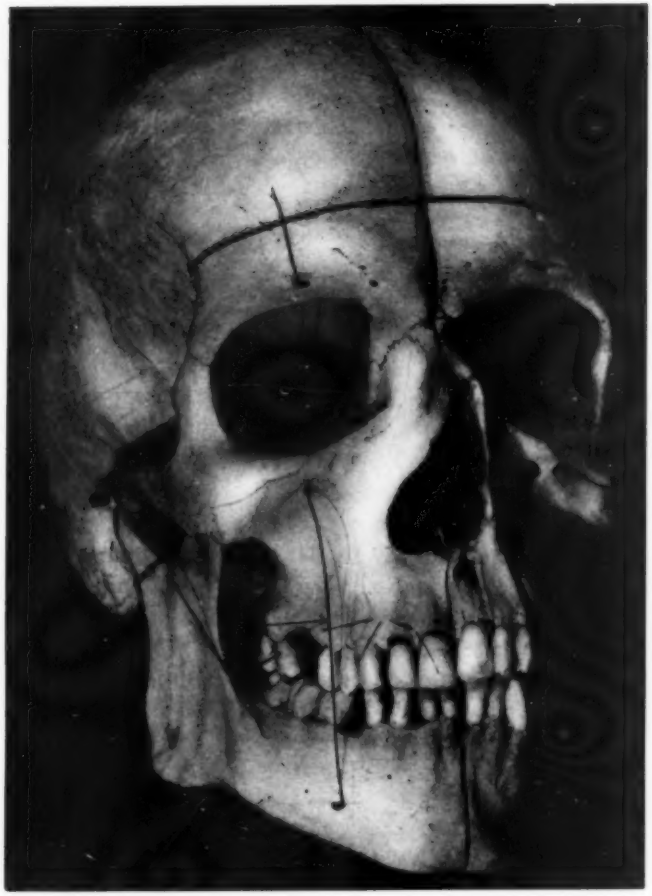

FIG. 1.

External view of skull showing mental foramen and sieve-like foramina at the alveolar border in the maxilla, important in infiltration anæsthesia. Note that the supra-orbital, infra-orbital, and mental foramina are in a direct line.

FIG. 2.

Internal view of skull: a bristle has been passed into the inferior dental foramen, and a line drawn marking the pterygo-mandibular fossa where the fluid is deposited in mandibular anæsthesia. Note also the internal oblique line and retro-molar fossa.

FIG. 3.

Method of holding the syringe for puncturing the mucous membrane in the injection for mandibular anæsthesia.

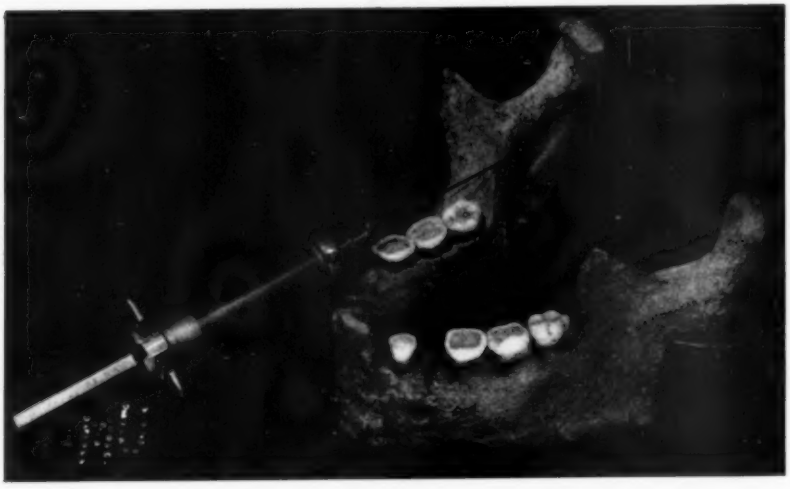

FIG. 4.

First position of the needle in injecting. The point of the needle should rest on the internal oblique line 1 cm. (½ in.) above the occlusal surface of the last molar tooth.

FIG. 5.

Final position of the needle which rests in the pterygo-mandibular fossa where the solution is discharged.

FIG. 6.

Position of syringe and needle in mental anæsthesia on the right side.

FIG. 7.

Radiograph of injecting for mandibular anæsthesia on the right side.

FIG. 8.

Radiograph of injecting for mandibular anæsthesia on the left side.

FIG. 9

Radiograph of injecting for mental anæsthesia on the left side.

DISCUSSION.

Mr. J. L. DUDLEY BUXTON: It might perhaps be of interest to cite my own experiences during the last two years. This method seems to me to be of particular value in dealing with war injuries—e.g., in cases when a minor operation is necessary, such as the removal of sequestra. My usual routine is to eliminate shock as far as possible in jaw cases—of which I have had some little experience—by the use of additional hypnotic drugs, as I am fully convinced that unless the operator can put out of his mind that he is dealing with a conscious person, even although he has satisfied himself that the analgesia is complete, he will not be able to perform a satisfactory operation. It is well known that, although a patient may feel absolutely nothing, yet he may say he is suffering agonies, and this should not be lost sight of. To prevent this I inject, subcutaneously, morphine tartrate $\frac{1}{4}$ gr., atropine sulphate $\frac{1}{100}$ gr. (scopolamine), hyoscine hydrobromide $\frac{1}{100}$ gr. In cases in which the patient is a healthy soldier one finds it necessary to give as much as $\frac{1}{4}$ gr. of morphia, in less healthy patients $\frac{1}{6}$ gr. being sufficient. The reason for employing all three drugs together is that, although they all have grave disadvantages when used separately, yet given together they counteract in some respects—fortunately the undesirable respects—and aid in others by bringing about the effect desired, namely, "a general feeling of don't care"; further, a good deal of drowsiness is obtained. A patient may help by holding bowls and by answering questions during the operation, but subsequently is unable to give a lucid account of it (amnesia). After these drugs have been injected the patient is allowed to go to sleep in a slightly darkened room where no one will disturb him. Half-an-hour before the operation the novocain injection is performed. Vomiting has been reported after the use of hyoscine hydrobromide (scopolamine) by many experienced medical men, but if used in conjunction with morphia and atropine I have never known emesis to follow, even when a general anæsthetic has been administered subsequently. This latter procedure would be unwise after such doses of S.M.A. (scopolamine, morphia, atropine), as although atropine stimulates the respiratory centre, yet there is a great tendency for respiration to cease should a deep narcosis be obtained. I have heard of one case in which inframandibular injection had been performed, where the patient, a middle-aged woman, suddenly collapsed ten minutes afterwards. She could only speak in a whisper when she had recovered her faculties. This condition lasted for about two hours, when finally she recovered her voice. There was no apparent sign of any organic disease of the heart; the patient complained of a feeling as of a piece of string wound tightly round her throat, which became looser if she breathed gently. It was subsequently discovered that there was a leaking joint between the needle and syringe which would have allowed a few drops of novocain to trickle down the throat; this apparently would not be enough to cause such alarming symptoms, but it seemed a possible explanation, as only 1 c.c. of a 2 per cent. solution of novocain was the dose administered. Even at the early

age of 6, children appear to be as good subjects for this method as adults, although one must bear in mind the different anatomical condition when injecting in the case of children. When there was some difficulty in obtaining novocain, I used beta-eucaine-lactate, employed in regional analgesia, though with only moderate success, although it is safe to use as high a percentage as 6 per cent. with this drug; but as a local analgesic the results were more favourable. Mr. Rowlett mentioned that unless novocain solution was freshly prepared from the tablets, it lost many of its valuable properties, and that it was unwise to use such a solution. About a year ago I had some ampoules of novocain sent me from Italy; I have used them just lately with success and without any trouble arising, but I certainly should hesitate to use novocain left over from the day before. The technique of this method requires to be fully mastered before success can be forthcoming, and above all things complete asepticity of instruments, solution, and hands is necessary. As regards alypin, in a case in which this drug was used in a very clean mouth, and with the utmost care to eliminate all septic dangers, pain with stiffness of the muscles and discomfort in swallowing occurred; this persisted for a fortnight from the time of the operation—the extraction of the second lower molar on the right side.

Mr. F. N. DOUBLEDAY: Mandibular anæsthesia was in frequent use during my period of study in Berlin eight years ago. Since then I have made little use of it on account of the fear of introducing sepsis too deeply into the neck as, for example, the region of the inferior dental foramen. I have often wondered what would happen if infection did occur, and have come to the conclusion that the best plan to overcome it would be the constant use of hot gargles to get the products of inflammation to the surface, perhaps as a post-pharyngeal abscess. During this war much use has been made of regional anæsthesia by Major Kazanjian at No. 20 General Hospital and by Mr. Dudley Buxton at King George Hospital, and the experience of these gentlemen confirms that of Mr. Rowlett that the risk of deep sepsis is small if proper precautions are taken. Before employing mandibular anæsthesia the German surgeons made a practice of giving their patients potassium bromide 15 minims.

The PRESIDENT: Before calling upon Mr. Rowlett to reply to the general discussion, may I ask him whether he aims at a penetration of the inferior dental nerve, and generally obtains it, or whether he is content with an injection of the cellular tissue in the immediate neighbourhood of the entry of the nerve into the mandibular canal? Mr. Rowlett has incidentally pointed out the liability of an undesirable and protracted anæsthetic effect to follow upon the use of a syringe which has been kept in methylated spirit, containing a drop of spirit at the moment of using. In corroboration, I am able to bear testimony to the value of injecting alcohol into the sheath of the inferior dental nerve as a means of relief and cure of severe trigeminal neuralgia affecting the area of supply by that branch, a cure which in one case has lasted for a period of at least two years to my knowledge.

Mr. A. E. ROWLETT (in reply): All authorities on local anæsthesia are strongly of the opinion that a freshly made solution is less toxic and has better anæsthetic qualities than a solution that has been kept for any length of time. Seidel declares that deterioration of the drugs begins to take place ten minutes after the solution is made. The pain and swelling after a correctly performed mandibular anæsthesia is very slight, and in a large number of cases it is entirely absent. I have obtained thoroughly good results enabling me to do permanent conservative work in a child aged 8. Complete anæsthesia takes place in all the teeth of the lower jaw in a period varying from twenty minutes to half an hour. A patient should be under supervision during that period. It is not intended to penetrate the inferior dental nerve, but to deposit the fluid in the pterygo-mandibular space around the nerve. Numbness of the lower lip on the same side as the injection, occurring about five minutes after injection, is a sure sign of a successful operation, and if this is not present success cannot be hoped for and the injection must be repeated.

FIG. 1.

FIG. 2.

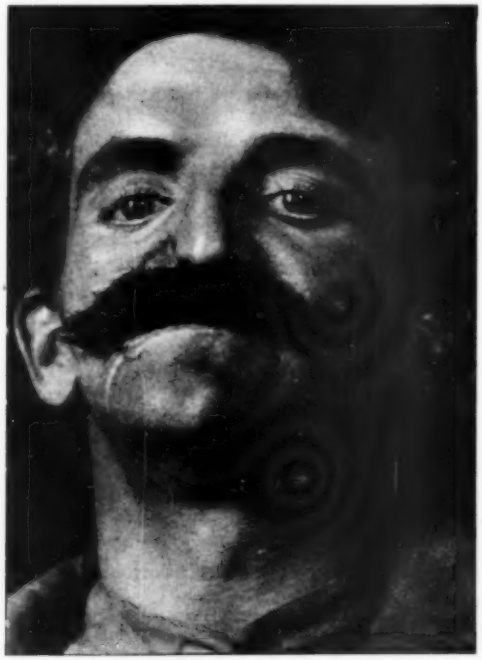

FIG. 3.

Illustrating Mr. J. Lewin Payne's case of facial restoration by means of a mechanical appliance (see pp. 17, 18).

Section of Odontology.

President—Mr. W. B. Paterson, F.R.C.S., L.D.S.Eng.

DEMONSTRATION OF CLINICAL CASES OF GUNSHOT INJURIES OF THE JAW AND FACE.

(*March* 26, 1917.)

Comminuted Fracture of the Mandible.

By G. Paton Pollitt.

This patient, aged 21, was wounded on the Somme on November 13, 1916, by either a rifle or machine-gun bullet, and was sent to Rouen, where no special dental treatment was undertaken. Nine days afterwards he was admitted to the King George Hospital, under Mr. Carson and Mr. Spokes—i.e., on November 22. I saw him on November 23. Radiographs were taken, and it was seen that he was suffering from a comminuted fracture of the mandible in the molar region on each side and at the symphysis (figs. 1 and 2). The wound of entry was on the left side in the molar region and the exit on the right; the injury of the soft tissues was not very great, but the mouth was very septic.

On November 24 he was given a general anæsthetic, and two roots of a lower first molar on the one side and the fractured first molar on the other were removed. An attempt was made under the anæsthetic to get an impression of all the lower teeth, but this was found impossible owing, first, to the large amount of swelling, and, secondly, to the fact that all the teeth with their attached portions of bone were so freely movable. Therefore, I took an impression later of the lower incisors and premolars and of the upper teeth (fig. 3). There was considerable displacement of the incisors and premolars; they were displaced downwards and inwards. The lower model was then divided and put in

proper alignment and a cap splint made for both upper and lower incisors and premolars—i.e., the molars beyond the area of fracture on either side were not included in the splint.

Both splints were cemented into place on December 12, the fracture at the symphysis being reduced at the same time. Both splints had hooks on the labial surface. The splints were wired the

FIG. 1.

Right side.

next day, the lower incisors being drawn up into fairly good articulation, the procedure being simple owing to the mobility of the parts. The molars in the mandible were not displaced much in relation to the upper teeth, so that by immobilizing the front teeth these were held more or less firmly in their right articulation. The splints came off once or twice and were recemented.

FIG. 2.
Left side.

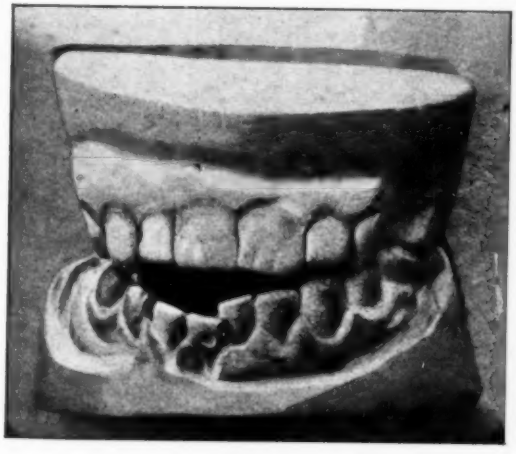

FIG. 3.

On February 19 the patient was sent to the Auxiliary Hospital and readmitted on March 2 for examination, when the splints were removed and radiographs taken (figs. 4 and 5).

I propose now to put on a cap splint covering all the lower teeth, with a partial cap for the upper front teeth merely as a rest for the

FIG. 4.

Right side.

bite; the articulation is quite good, and with a little grinding of the molars later will be practically normal. The fracture at the symphysis is quite firm, the right central incisor being dead. There

is no suppuration. In the molar region on the left the solidification is very good also, and there is no suppuration, but there is slight movement on the right side and slight external suppuration.

I think that the interest in the case lies in the amount of solidification that has taken place during the three months that he has been

FIG. 5.
Left side.

under treatment; and it helps to show us that one must not be in too great a hurry to extract teeth in a line of fracture or implicated in the comminuted areas. In this case had I wished to take out the teeth implicated in the fracture, it would have meant the extraction of a large

number, as there was a definite transverse fracture in the alveolus, in the region of the left incisors and premolars, these teeth, with the alveolus, being extremely loose.

In spite of his being wired up, and of the sepsis in the mouth, the patient has done extremely well, and we were able to keep his mouth sufficiently clean. He was unwired now and then and the mouth cleaned and swabbed with 2 per cent. of iodine, and the opportunity was also taken of doing this when the splints came off.

The points to which I wish to draw attention are:—

(1) The fact that the molars were not included in the splints.

(2) That practically no teeth were extracted as being included in the fractured area, because they were most of them implicated.

(3) The amount of new bone formation during the three months that the splints were *in situ*.

(*March* 26, 1917.)

Report on Treatment of a Case of Shrapnel Wound of the Mandible, in which Bony Union was re-established after great loss of Substance.

By B. Maxwell Stephens.

The treatment of this case, originally, was carried out in conjunction with Mr. Cyril Nitch.

The patient, a Canadian, aged 19, was wounded on October 12, 1916, and received on October 23, at the Facial Hospital, Brook Street, London. He was then in a marked state of collapse. There was a small wound in his right cheek and a larger one in his left; the anterior portion of the mandible hung loosely forward so that only with great difficulty could he close his lips; speech was impossible for several days. Fragments of shrapnel had struck the lower border of the mandible on the right side, entered the mouth, ploughing through the sub-lingual region and emerging through the left cheek just anterior to the opening of the parotid duct. On the right side immediately anterior to the first bicuspid a line of fracture ran obliquely downwards. Above the symphysis menti there lay an extensive area of comminution,

bounded on the left by a second line of fracture in the bicuspid region; beyond that, just anterior to the second molar, was situated a third line of fracture. The teeth between the right lower second bicuspid and the left lower second molar with the alveolus which supported them were missing. All the teeth in the upper jaw were present and uninjured. The mouth was cleansed and swabbed with tincture of iodine and a tongue scraper used. Irrigation with saline solution from a portable pneumatic tank was effected every two hours in the day and as the patient at first slept badly, at the same intervals during the night. In three days the condition of the man had considerably improved.

On October 27, under a general anæsthetic, a sequestrum was removed from the neighbourhood of the fracture on the left and pendant drainage established in the median line of the mouth; through the same incision also as that made for the drainage, was passed a second and longer tube, the extremities of which were attached to a rod maintained vertically before the mouth. This rod was held in the middle line by a horizontal bar attached to ear pieces suspended from a head cap. By this means an attempt was made to support the loosely hanging portion of the jaw.

On November 8 secondary hæmorrhage occurred; in searching for the source it became necessary to separate and remove the largest fragment of bone lying between the lines of fracture. The possibility of its union occurring with the body of the ramus on the right was indicated by a large mass of callus which had been deposited at that extremity of the fragment. The fragment removed measured 47 mm. along the inferior border and 15 mm. in depth. Control of the hæmorrhage was obtained by suturing a lint pad within to two short lengths of rubber tubing placed against the skin. After thirty-six hours the pad was removed and no hæmorrhage recurred. It is worth noting that the patient subsequently exhibited a marked hæmorrhagic tendency on more than one occasion. After the removal of this remaining fragment from the fracture, the rami swung towards each other, crowding on to the tongue; on the left side the lower second molar was therefore wired to the corresponding tooth in the maxilla, and on the right side a small intermaxillary wire splint was fixed.

On December 6 a permanent splint was fixed; its form can be followed in the second radiogram (fig. 2). From either side bars of German silver, rectangular in section, made in two halves, spring from attachments fixed to the remaining teeth. These bars are carried round the line of the alveolus till they meet and considerably overlap. When the

attachments had been fixed in position the opposite sides of the mandible were moved apart from each other and the bars wired firmly together in that position. At the end of a week callus was found to be depositing; the wiring was then removed and the parts forced still further outwards;

FIG. 1.

Skiagram taken by Mr. C. A. Clark on November 20, 1916. The interfragmental region is shown. The mandibular teeth are temporarily secured to those in the maxilla.

effective occlusion having been secured between the upper and lower teeth, the bars were rewired in the new position. At the same time a removable vulcanite pad was attached to the bars to stretch the scar in

the left cheek and prevent the lip falling in. It is convenient to mention here that to avoid the formation of a depressed scar, massage was applied daily to the cheek for two months. The deposit of callus steadily continued. A small sinus however was found to have persisted in a position situated anteriorly and lingually to the molar on the left

FIG. 2.

Skiagram taken on March 22, 1917, by Mr. C. A. Clark. Density of callus very slight. Form of splint is shown.

side. When explored it was found to lead directly downwards to the interfragmental area, but no cause for its persistence could be located.

On March 22, therefore, further stereoscopic radiograms were taken;

one of the pair is reproduced herewith (fig. 2), and shows that the callus has bridged the space entirely except at one spot on the left side in the neighbourhood of the sinus. The foregoing is the history of the case up to date. As the apical condition of the molar on

FIG. 3.

Skiagram taken on May 15, 1917. Bony union has been established.

the left now appears to be doubtful, it has practically been decided to remove it; before this is done, however, it is felt that it will be wise to further explore the sinus under local anæsthesia.

Note.—Since reporting the above, I have fortunately been able to find the apex of a tooth in the sinus above referred to; the molar tooth has therefore been spared. The bony union which has now been secured I consider has been helped by a daily application of high frequency electric current. The splints are now off and a heavy tin denture has been made and inserted. The man masticates well and will be recommended for active service again.

(*March* 26, 1917.)

Cases of Gunshot Injury of the Face and Jaw, with Special Reference to Treatment.

By F. N. Doubleday.

The first of my cases is that of an Australian, Private G. He had an extensive loss of tissue of the left cheek. This was successfully dealt with by a plastic operation. After the operation a dense mass of scar tissue formed in the cheek, which prevented him from opening his mouth and greatly hindered the treatment of his jaw injury. In order to soften his scar and render it more pliable he has been treated for three months with Dowsing heat and, as those of you who have examined him will have observed, the scar is now fairly supple, and he can open his mouth freely. The heat is applied for fifteen minutes daily, a 50-candle power lamp being placed about 6 in. from the patient's face and the rays interrupted by the hand of the nurse being passed to and fro between the lamp and the patient's face. This treatment we have employed for several cases where excision of the scar was, for various reasons, undesirable, and always with most satisfactory results.

I will now give you a brief account of the other cases which you have already examined in the clinical room. The first is that of Private S., who was wounded on July 13, 1916, and admitted to King George Hospital on July 18, 1916. On admission the patient had a wound of entry just below the zygoma on the left side; the bullet had travelled down towards the chin, fracturing the maxilla and mandible. There was a large open, septic wound in the region of the chin on the left side and an open wound through into the mouth. Part of the

mandible in the left canine region was missing; the left fragment was internal to, and overlapped by, the right fragment to the extent of nearly an inch. There was also a fracture of the right horizontal ramus of the mandible and a wound of the left hand. The following teeth were remaining:—

8	8
8 4 3 2 1	2 3 4 5

The lower left incisor and a piece of shell were found between the fragments when an anæsthetic was administered. The skiagraphic report showed that there was a double fracture in the canine region extending back into the premolar region upon the left side. At first an attempt was made to retain the lower front teeth and, by applying a splint to them, to reduce the displacement of the bone. But the persistent suppuration and the failure of the teeth to tighten up made me feel that they were only hindering the treatment of the case, so that on August 18 these teeth were extracted with great reluctance and considerable fear as to the ultimate result, from total loss of any support for a splint. As the fracture was not yet united I feared that the displacement would recur, but this did not prove to be the case: a good deal of necrosis went on, and the sinuses were reopened and scraped on August 18 and again on September 5 and 17, but by the end of September it was evident that firm union was beginning to occur. As late as January 27, 1917, some small spicules of bone were exfoliating, but at the present time the patient has a firm bony union of his jaw without any other deformity than that of a rather flat floor to his mouth.

The second case, that of Private L., is of a similar but more severe character, as will be seen from the diagrams (figs. 1 and 2). There were several fissured fractures running across the maxilla, and at least four fractures in the mandible. Almost all the teeth were standing, but were loose and moving about, both with the fragment and within their attachment to it. Here, as in the first case, I was reluctantly driven to extract all the teeth, fearing all the time that serious displacement would ensue which it would be difficult or impossible to overcome when there were no teeth from which anchorage could be secured. Fortunately, however, this fear proved to be groundless, for, as those of you who have examined the patient will have found, there is perfectly good bony union and no displacement.

In both these cases the removal of the teeth was so extensive as to render the use of a splint impossible. Any temporizing measures would

have been useless owing to the extensive nature of the injuries. Yet in both cases, as you have seen for yourselves, perfectly satisfactory bony union has ensued without displacement.

The third case is that of Private K., who was wounded on August 20, 1916, and admitted to King George Hospital on September 3, 1916. On admission he was found to have a fracture on the left side of the mandible in the region of the first molar; there was an external wound discharging pus freely. There was a scalp wound in the left parietal

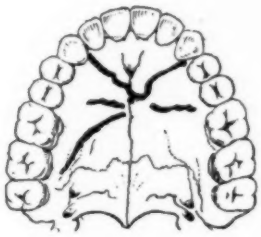

FIG. 1.

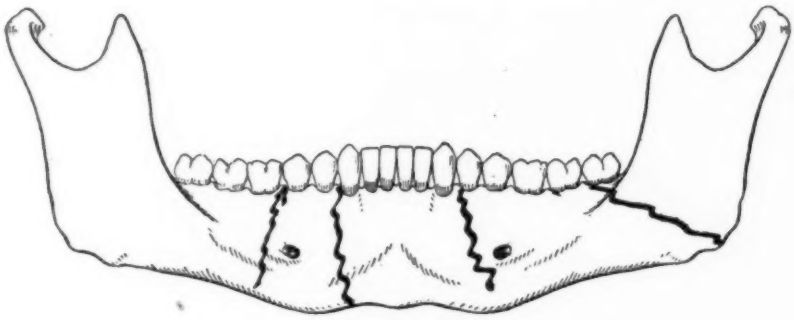

FIG. 2.

region going down to the bone. A double wire splint united by ligatures had been inserted in France. There was at first, as is so usual in these cases, a good deal of discharge from the wound and some necrosis. The case dragged on, and by December no bony union had occurred. The wire splints were therefore removed, the second premolar and first molar on the left side were removed, and a double cap splint, united by wiring, inserted. At the same time the external sinuses were opened and scraped. Within six weeks firm bony union

had occurred, and all discharge had ceased; a pretty clear indication as to what should have been the line of treatment from the beginning.

The fourth case, that of Private O., is of interest because there arose the question of the amount of tissue which can be bridged over by new bone. The patient was wounded on July 14, 1916, by shrapnel; nearly an inch of the entire depth of the bone in the region of the symphysis was shot away (figs. 3 and 4). The patient was admitted to King George Hospital on July 21. There was then so much swelling and discharge of pus that the only treatment adopted was to provide free drainage and to relieve the patient's pain by wiring the upper and lower teeth together. By August 7 he had improved sufficiently to have a cap splint applied to the remaining teeth—the canine and two premolars upon the right side and the canine and first premolar upon the left side. The halves of the mandible showed a good deal of tendency to fall in towards the middle line; owing to the unopposed action of the external and internal pterygoid and the mylo-hyoid muscles there was a considerable tendency at first for the splint to work off. By December there was some bone along what had been the inferior margin of the mandible, but the union was not firm enough to allow any pressure to be put upon it and the two halves of the jaw were movable upon each other with firm pressure. The splints were reapplied and at the present time, six months after the injury, I believe you will allow that the patient has a good firm bridge of bone formed across the gap and that there is no apparent deformity. With the help of a denture I believe that this patient will shortly be able to masticate hard food quite well.

The points that I wish to raise in connexion with these cases are as follows: First, the value of radiant heat as a means of making scar tissue supple; it appears to me to be a simple and effective method of treatment. Secondly, in these cases, where very extensive comminuted fractures have resulted from gunshot injuries, the satisfactory results which have followed the extensive extraction of teeth. Thirdly, as to the best type of splint for use in these gunshot injuries of the jaws. Personally I believe that the cap splint is by far the most satisfactory appliance. In most cases I use a double cap splint, cemented to the upper and lower teeth with oxyphosphate of copper cement; the copper is an excellent antiseptic. To these splints stout hooks are attached and the jaws are immobilized by wiring them together with brass wire. By this means absolute immobility is secured, and if it is desired to open the mouth to clean it, or to ascertain the progress of the case, it is easily done by cutting the brass wire and then rewiring after the examination

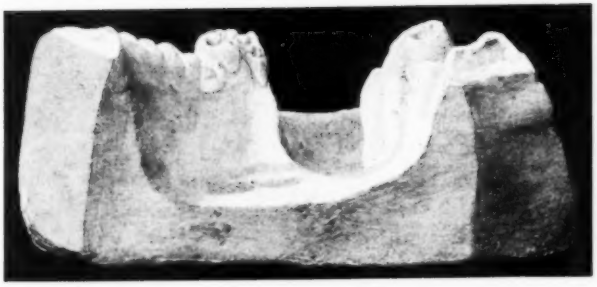

FIG. 3.

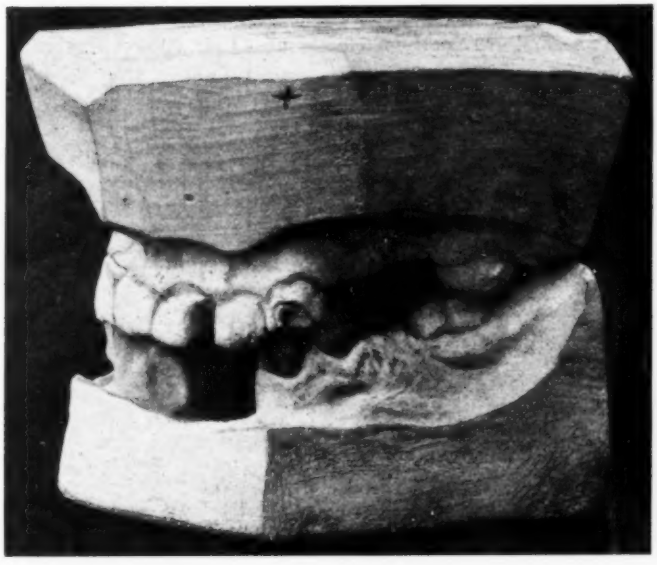

FIG. 4.

is complete. This type of splint seems to me simple, clean, and efficient. Fourthly, what are the essentials of treatment? I believe that drainage comes first and immobilization of the jaws second. By drainage I mean opening up and scraping the sinuses from time to time, much as one would treat a septic tooth socket which refuses to granulate from the bottom. By this means we encourage the separation of small sequestra, but do not run the risk of removing larger pieces of bone which may be sufficiently nourished by the serum, or by flaps of periosteum to live and form new centres for osteogenesis. I cannot too strongly deprecate the opening and scraping of the wound and the removal of bone on an extensive scale by the surgeon. If this means of treatment had been adopted in Case IV the patient would certainly not have obtained such a good jaw as he now has. In the early stages of the case it is impossible to tell which fragments of bone will live and which will die. The apparent presence of loose bone in the skiagram means nothing; to remove it by operation is disastrous; the only safe procedure is to wait for it to separate and exfoliate and then to remove it six or eight weeks after the injury. The next essential appears to me to be to promote immobility of the jaws and so to secure absolute rest. These soldier patients are strong hearty fellows, and to tell them not to use their jaws for hard food is useless unless they are prevented from doing so by the fixity of the splint, and I believe that free drainage followed by a period of absolute rest secures the most speedy and satisfactory results.

CORRIGENDUM. — On p. 23, line 14 (Rowlett, "Mandibular Anæsthesia"), read "8 mgr." instead of "8 grm."

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

J. Y. W. MacALISTER

UNDER THE DIRECTION OF

THE EDITORIAL COMMITTEE

VOLUME THE TENTH

SESSION 1916-17

SECTION OF OPHTHALMOLOGY

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1917

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SECTION OF OPHTHALMOLOGY.

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Section of Ophthalmology.

President—Mr. William Lang, F.R.C.S.

(*November* 1, 1916.)

Congenital Pigmentation of the Optic Disk.

By G. Winfield Roll, F.R.C.S.

An elderly police pensioner consulted me at the Royal Westminster Ophthalmic Hospital at the instance of Dr. W. Evans. He is the subject of glycosuria and has well-marked retinitis diabetica in the right eye, the vision of which is defective, owing to macular exudates and hæmorrhages. The left eye has good visual acuity, $\frac{6}{9}$; the optic disk is normal in outline and arrangement of retinal vessels; its colour is uniformly inky black, forming a remarkable contrast to the surrounding fundus.

I believe the condition to be congenital owing to the even distribution of the tint of the optic disk. A similar appearance of an optic disk has never hitherto come under my observation; a few instances of a somewhat similar condition are on record. I exhibit a sketch in colour of the abnormality.

(*November* 1, 1916.)

Pigmented Plaque in Corneo-scleral Junction; ? Nature.

By Leslie Paton, F.R.C.S.

Patient is a girl, aged 7. The condition was noticed first by her mother about seven weeks ago. It consists of a small pigmented area at the junction of cornea and sclera of left eye, about 2 mm. in

diameter and brown in colour, with a slightly darker centre. The fibres of the iris in the sector corresponding are slightly pushed forward as if by underlying growth. When the pupil is dilated, however, no definite growth can be seen.

I have brought this case up for the opinion of the Section, because I have not previously seen a case exactly like it. I shall be glad if anyone can throw light on its nature. The mother is quite sure there has been no injury.

DISCUSSION.

Mr. STEPHEN MAYOU: In the centre of the black part of the mass there is a little grey spot, and it looks as if the iris fibres were drawn forward to it and adherent to the back of the cornea. If I had seen this case and did not know the history, I should have said that the child had had a perforating injury there, and that, in consequence, the iris had become adherent, and that the black mass was uveal pigment in the scar. There seems to be no history of injury, but in the case of small children this does not count, as frequently they do not come till several days after such an injury, as there is usually little pain and they are afraid of getting themselves or their friends into trouble.

Mr. LESLIE PATON (in reply): No skiagram has been taken, and I could not transilluminate owing to the lachrymose condition of the child. It is only a week since I first saw her. At first sight the obvious thing was to suppose that something had been driven through into it, because there is a darker centre to it. But there is no history of injury at all. It was first noticed seven weeks ago, and the child was not in pain, and has never had pain. The mother is certain that if the mass had been there before she would have noticed it. The iris fibres bulge sideways when the pupil is small. Of course the pupil has not yet quite recovered from the effect of the atropine, but I could see the iris fibres curled apart one from another, and that made me suspect there might be a growth in the ciliary region and that what we see might be a secondary pigmented outgrowth from the ciliary region. On the other hand, on looking at it very carefully, its position in the corneal tissues seems rather too superficial for that to be the case. I have not inspected it with a binocular microscope. It may be subepithelial in the cornea.[1]

[1] *Note.*—January, 1917: I have since seen a case of an exactly similar nature—i.e., a brown pigmented plaque at the margin of the cornea, resulting from a needling for lamellar cataract performed about ten years ago. Despite the mother's absolute denial of any accident in the case shown, this, in my opinion, renders it most probable that also it is the result of a needle piercing the cornea.

(*November* 1, 1916.)

Case of Pigmented Degeneration of the Retina, associated with Epileptic Fits.

By F. E. BATTEN, M.D.

THIS is a case of Captain Ormond's, and was referred to me for an investigation of the nervous system. The patient is a man, aged 23, and he first noticed failure of eyesight in 1910. This steadily progressed, and has done so more rapidly during the past year. Epileptic fits began in 1913, and these have become more frequent during the past year. Captain Ormond found there was a central scotoma in both eyes, somewhat to the temporal side, and optic atrophy, with some pigmentary disturbance at and near the macula with diminution of vision to $\frac{6}{60}$.

Physical examination of the nervous system had a negative result. Both the blood and the cerebrospinal fluid have been examined, and give a negative Wassermann reaction. The father undoubtedly suffered from syphilis, but no evidence of the disease is present in the son.

The case is shown in order to elicit the opinions of members of this Section with regard to the pigmentary change which is present, as to which class of degeneration it belongs. The patient is the fourth member of the family, and no other member of the family is affected, so far as I know. His brother is a prisoner in Germany; his two sisters, who are older, I have not been able to see, but they are said to be normal.

DISCUSSION.

The PRESIDENT: I think the scotoma involved the fixation point in one field but not in the other. The scotomata did not appear to be quite symmetrical. The absence of a positive Wassermann test seems to be against it being due to congenital syphilis: it was negative both in the blood and the cerebrospinal fluid. I think this is the type of case which might profitably be brought up again after further observation, to see what happens to the disks. They struck me as still being congested.

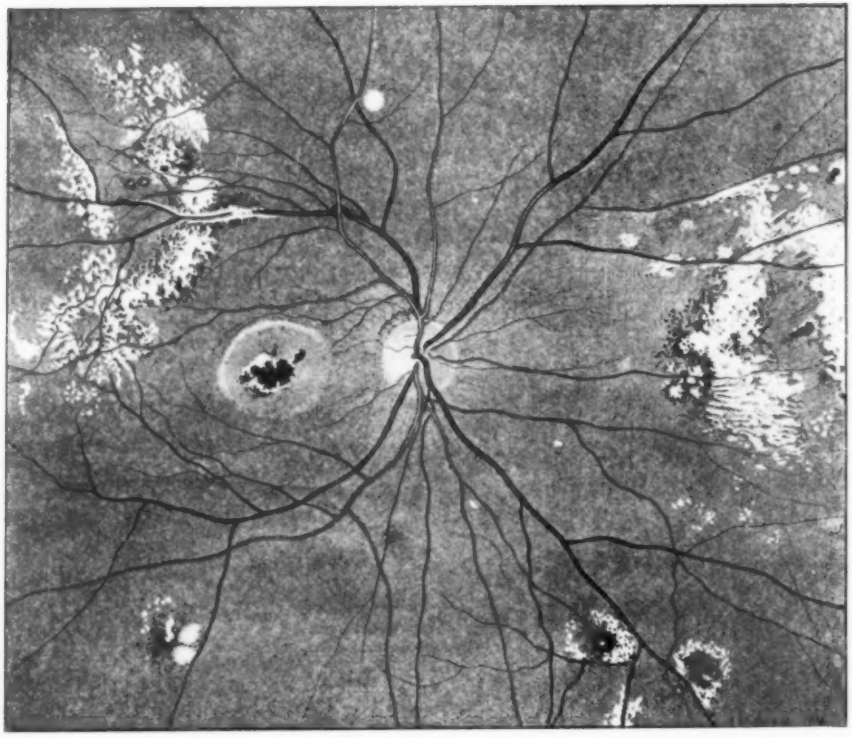
Captain ORMOND: This patient was sent to me first of all because the sight was failing rapidly, and I elicited from his mother the fact that he had had a number of fits. The first fit was in October, 1913. He had another the

following month. He had two in 1914, and five last year. Since then he has had seven fits. They are increasing in frequency. The disks are very hazy, and the vessels contracted. The pigmentary changes in the macula spread a good way towards the periphery. At first, owing to the marked history of syphilis in the family, I thought that we were dealing with a case of congenital syphilis. His teeth, however, showed no signs of that disease. Dr. Gordon Goodhart did a Wassermann reaction for me, which was negative. Thinking the patient might have some other cerebral condition, I sent him to Dr. Batten, who investigated the case very thoroughly. Mr. Paton has since taken his fields carefully, during his stay in Queen Square. The patient has also had some extraordinary sores on his legs, the nature of which I do not know. He had about half-a-dozen of them, and they were a long time healing. The sight is certainly failing now, and has been so for the last eighteen months. He can fix with both eyes, if the object is sufficiently large. The scotomata are not absolutely symmetrical.

Mr. RAYNER D. BATTEN: I think the macular type of degeneration, having developed, does not progress, but remains stationary. This case appears to be progressive, which points to it being a syphilitic type of central retinitis, rather than belonging to the macular type.

Mr. STEPHEN MAYOU: With regard to the stippled appearance in the retina, I think most of the cases show this, in addition to the macular changes. I have seen altogether four families with the disease: two have been shown here, and two are represented in patients I have at present under my care: they all show stippling at the periphery of the retina. In the two families of my own, which I have mentioned but not shown because I was not quite sure they belonged to this class, one could not say there was definite cerebral degeneration at all: all one could say was that they were rather curious people, of not quite normal mentality. Otherwise, the appearance in the fundus was exactly similar to that in this case. They were two sisters in one family, and father and son in another, and in none was the Wassermann test positive.

Mr. J. HERBERT PARSONS: There does not seem to be much evidence that this case belongs to the group named. I understood Dr. Batten to say that he found nothing wrong with the central nervous system; except for the history of epilepsy it seemed to be normal. It is well known that there are seldom changes in the fundus in epilepsy; and it is a question whether it is not a mere coincidence that this condition of eye should occur in a patient who has epilepsy. I should very much like to hear the opinion of the senior members of the Section as to the frequency of macular changes in congenital syphilis, similar to those in this case. My own experience is not sufficiently great for me to give an opinion. I should have thought it was not very rare to find changes in the macula similar to these. I agree with Dr. Batten that the cases which he has grouped together are cognate. As I have already said more than once, I think the amaurotic family idiocy cases should be kept abso-



FLEMING:
Case of Retinal Arterial Disease, with Miliary Aneurysms and Exudates.

lutely distinct, for the other cases are only remotely related; and in this case the evidence of association seems to be particularly slight.

Mr. LESLIE PATON: Dr. Batten asked me to see this patient when he was admitted for observation at the hospital. Before I knew anything of the history, I was struck with the extreme resemblance to one of the cases shown by Dr. Batten which I saw two years ago, an instance of Batten maculo-cerebral degeneration. On further examination I found that there was an absolute central scotoma 10 mm. wide, extending for some distance out, in both eyes. We took his fields with the Bjerrum screen. There was another feature, namely, that he complained of night-blindness, which had been experienced some considerable time. I am surprised to find that there is no obvious limitation of the peripheral fields. The whole retina has an atrophic appearance: it has the "autumn-leaf" appearance of retinitis pigmentosa; there is certainly a generalized atrophy of the retina, and there are the very much reduced blood-vessels which we see in retinitis pigmentosa. When I learned the history, I thought it might be retinitis of syphilitic origin, with subsequent atrophy. But I think syphilitic retinitis, as distinct from choroido-retinitis, is rare in congenital syphilis. It is commoner in association with acquired syphilis. In view of the negative Wassermann tests, I feel myself driven to the view that this case is allied to the Batten type of cerebro-macular degeneration. I have already expressed my view that these cases should not be mixed up with the ordinary Tay-Sachs type of amaurotic family idiocy; but I know that Dr. Batten and I do not quite agree about that. There is little doubt about the failure of vision in this case in 1910. He was seen by somebody then, but it may have been simply a question of an error of refraction necessitating glasses.

(*November* 1, 1916.)

Case of Retinal Arterial Disease, with Miliary Aneurysms and Exudates.

By NORMAN B. B. FLEMING, Lieut. R.A.M.C., M.B., Ch.B.

C. came under my notice at the Central Recruiting Depot, and I found that he had a complete central scotoma in his right eye. The patient had been aware of this for some time, but had on no occasion sought any advice. His mother attributes the loss of vision to a blow on the eye in childhood, but medical attention was not required and the injury apparently was not serious; he complains of no other sign or symptom. The fundus presents widespread changes resembling those found in retinitis circinata, but not showing the

characteristic distribution. There is a dense patch of pigment at the macula, surrounded by an area of light colour, this in turn having a definite limit which is nearly white and which at the upper part appears to be raised. There are very extensive opaque white patches in nearly all parts of the fundus, and the retinal vessels are in some cases embedded in this material. Several old hæmorrhages are present and a few recent hæmorrhages at the periphery. There is some local sclerosis and aneurysmal dilatation of the retinal arteries and thickening of the veins, particularly the superior temporal vein.

In a collection of cases of retinitis circinata Fuchs[1] reports that in one case only was alteration of a retinal artery found, and I have been unable to find any other case in which such change has been noted. The pigment at the macula and the irregularity and extensiveness of the opaque white exudate are of a nature quite different from that described in any of the articles I have consulted.

(*November* 1, 1916.)

Speculum for Use in the Operation of Evisceration of the Sclera, and in Enucleation of the Eyeball.

By A. C. Hudson, M.D., F.R.C.S.

The instrument I have exhibited is designed on the model of a modified trumpet-shaped ear speculum. It is intended for use in the operation of evisceration of the eyeball, and in some cases of enucleation.

In the former operation it affords a perfect view of the region of the optic nerve entrance, the pressure of the leading edge of the instrument

Speculum (two-thirds actual size.)

[1] Fuchs, *Rev. Gen. d'Opthal.*, 1894, xii p. 76.

ensuring a bloodless field, so that all remnants of the choroid adherent around the nerve head can be removed with certainty.

In the case of accidental button-holing of the sclera in the course of enucleation the residual stump can be easily identified by means of the speculum, and any portion of the globe that remains adherent to the optic nerve can be readily demonstrated and removed.

I hope that the use of this instrument may serve to diminish the incidence of cases of imperfect removal of the choroid in modified excision of the eyeball, with its attendant risk of sympathetic inflammation, several of which cases I have myself recently met with.

(*November* 1, 1916.)

The Photography of Macroscopic Eye Specimens.

By Lieut.-Colonel and Mrs. R. H. Elliot.

The advantage of being able to photograph one's own specimens of pathological eyes, whether macroscopic or microscopic, is too obvious to call for comment. Any worker who wishes to do this will find himself confronted with considerable difficulty in obtaining information as to details of technique, exposure, &c. In photography detail is everything. We have recently had occasion to photograph a number of both naked-eye and microscopic sections of eyes, and the object of this paper is to place some of the information so gained at the disposal of others. It is not proposed to waste space over a description of methods of sectioning eyes, &c. This has already been done elsewhere. We start, therefore, with the half-eye, or with the mounted section ready to hand.

Method of Photographing Cut Eyes.

If a good photograph of a sectioned eye is to be obtained, attention to certain points is essential: (1) The eye must lie completely under water; (2) the source of light must be good and even; (3) the camera must be placed vertically above the object in order to avoid reflexes; (4) the object photographed must be so placed that its image will occupy the centre of the plate, and adjustment should be available to secure this end with a minimum of inconvenience; (5) a simple

arrangement is necessary to fix the eye in position during the whole period of exposure; (6) to save unnecessary retouching, the object should be photographed lying upon a dark and even surface, so that the background should in no way disturb the attention of anyone looking at the picture; (7) a suitable camera should be selected; (8) the question of exposure must be very carefully studied. We will take these points seriatim.

(1) It is most important that *the object photographed should lie completely under water*, and as near the surface of the fluid as possible. If any part of the specimen projects, the water immediately around it is drawn up towards it in a slope presenting a meniscus. This causes the appearance of a very embarrassing light reflex, not only detracting from the scientific value of the representation, but also completely spoiling the picture from an artistic point of view. It might be suggested that the object should be photographed either in a specimen bottle, or in a cup covered by a slip of glass. Experience has led us definitely to discard any such method. The best results are obtained when the intervention of glass sheets is completely avoided. There is, however, one point to bear in mind—viz., that it is necessary to be careful to see that no specks of dust settle on the surface of the fluid, as these will appear enlarged in the negative, and enormously so in a lantern picture obtained from a slide made from it. The dust in the room must not be disturbed beforehand, and any specks which settle must be carefully removed just before the exposure is made. This can best be done by means of a pair of forceps, or some similar instrument. Cotton and other fabrics should not be used, as they deposit particles in return.

(2) *The Source of Light must be Good and Even.*—It is possible to work either by daylight or by artificial light. Each has its own advantages. If daylight be used, it is advisable to work opposite a high window with the light coming from above, as in this way reflexes are more easily avoided. Daylight is more readily obtained and costs nothing, while artificial light is more constant, and possibly admits of a greater command of the illumination. This is said to be an advantage when it is desired to select for reproduction certain deeply seated parts of the specimen, such as a scar just behind the ciliary body. The best form of artificial light is that obtained from an arc lamp. Care must be exercised to prevent the rays impinging directly on the lens of the camera, or fogging will result. Satisfactory arc lamps are rather expensive, and their employment involves the use of a resistance.

A knowledge of them is necessary, but they are easy enough to work with after a little practice. They possess three great advantages: (1) The time exposure for them is constant, whereas when working with daylight, very considerable allowance has to be made for the season of the year, and for the nature and time of day. (2) The exposure is considerably shorter. The most convenient method of working the lamp is to have it placed sliding on a vertical bar set in a heavy stand, and so arranged that it can be clamped at any desired height. (3) We are able to eliminate the inconvenience caused by the shadows which we get when working with daylight.

(3) *The Camera must be placed vertically above the Object.*—In order to do this, some form of apparatus is necessary. The firms that specialize in such work will supply a very elaborate stand for the purpose. We found, however, that a comparatively simple arrangement in metal satisfied all our requirements. It was made for us by Messrs. Spiller for a very reasonable sum. The necessary parts of it are: (*a*) an arrangement to clamp the camera and keep it rigidly in position; (*b*) a framework to connect this with the floor or table; and (*c*) a simple device to carry the specimen in its tray, and to admit of the latter being raised or lowered to a suitable focus. In order to avoid reflexes, the tray should be made of wood or metal painted dull black, and not of glass.

(4) *The Centring of the Image on the Plate.*—This is a most important step. It can be carried out by one or another of a variety of mechanical arrangements, but in our experience it is far safer to trust to the focusing screen.

(5) *An Arrangement to fix the Eye in Position.*—We may clear the ground by laying down what our experience has shown us are mistakes. First, *no* fabric should be used. We have tried paper, cloth and velvet, and have discarded them all, as they deposit fluff on the surface of the water, and so tend to damage the photograph. Next, receptacles made out of millinery wire, though very nice in some ways, are hard to hide from the camera, and are apt to be unsteady. We think that the best arrangement is that of a metal plate, painted dull black, and cut so as to fit into the sides of a photographic developing tray. In this plate should be cut three holes to accommodate large, medium and small eyes respectively. These must not be too close together, to prevent inclusion of an empty one on the plate. It is advisable to avoid shaking of the object by rough movements in the room during exposure, as its position may thereby easily be altered. Nothing should be done that is likely to stir up dust.

(6) Much after trouble is saved both for prints and for lantern slides by paying careful attention to the question of *background*. Nothing can be better than a metal plate evenly painted a dull black as above suggested.

(7) *The Selection of a Camera.*—Practically speaking there would appear to be three methods open to the photographer. He may use a telephoto lens, a double extension camera, or an ordinary camera with the addition of a lens on the planiscope principle. Our own work has been done with a Goerz five-magnifying power telephoto lens, with ring attachment. We need hardly say that we bought it before the War. Ross now makes a similar instrument. The great advantage of a telephoto lens is that a picture can be obtained double the size of the object. The disadvantages are: (1) the cost of such lenses, and (2) the increase in the length of the exposure necessary. There is a prevalent idea that expensive lenses enable one to obtain better pictures. This is a delusion. The advantage of an expensive lens in ordinary photography is that it enables the operator to obtain a perfect picture with a very short exposure. This is due to the fact that the better lenses will give sharp and even focus with wide open stops, which the cheap lenses cannot do. If the exposure is to be a long one, a cheap lens will do as good work, within certain limits, as a very expensive one. If a double extension arrangement is used, or if an attachment, on the planiscope principle, is employed, the picture can be made life size. It can be subsequently enlarged if desired. The latter procedure is not quite so simple as it sounds, and in photography as in eye-surgery, the multiplication of operations is fraught with danger. The added expense of an enlarging lantern has also to be considered. There can be no question that a study of pathological changes in the eye picture is greatly facilitated by its magnification to two diameters. A small point which should be mentioned on the other side, is, that the distance at which the double extension, or the planiscope fitted camera is worked, is much less than that required when using a telephoto lens. This makes the stand required less clumsy, and the working proportionately easier.

(8) *The Question of Exposure.*—This is far and away the most important point we have to consider, but no hard and fast lines can be laid down, since so much will depend on the source and nature of the light, on the brand of plate, and on the stop used. There are, however, certain points which require consideration. The fact that the photographs are taken under water is of very great importance, for the exposure needed is much greater than that for dry objects, indeed it is

ten times as much. We have also formed the opinion that the shallower the layer of supernatant water, the less the light is interfered with photo-chemically. On this subject we do not venture to dogmatize, as we have not worked it out. Another point to be borne in mind is that the majority of sectioned eyes are very dark objects, owing to the presence of the uveal pigment. This lengthens the exposure and also makes it extremely difficult to bring out the appearance of parts which lie in the shadows caused by the source of illumination. Yet another point is, that inasmuch as we are photographing hollow objects, the details of which do not lie in one plane, we must necessarily use comparatively small stops (F16, or thereabouts, with an ordinary lens); this again adds to the length of exposure. Our experience with the telephoto lens in winter time, under not very brilliant conditions of lighting, and using F11 and rapid plates, was that an exposure of from twenty minutes to half an hour was necessary. With a double extension camera, or with a planiscope, the period would be greatly lessened, and probably would be nearer two minutes. Each worker must find out these details for the conditions under which he works, but we think it is as well for him to err on the side of long exposures at first.

Closely allied to this question is that of the plates to be selected. We worked with Imperial Non-filter plates, but those working with cameras which require a less exposure might find it an advantage to use slower plates.

Magic Lantern Slide-making.

There are two methods of making magic lantern slides—(1) by contact, and (2) by the use of a camera. The former method is by far the simplest, as all that it is necessary to do is to place the plate in contact with the negative in a printing frame and to expose it to a chosen source of light for a definite time. The limitation of this method is that the magnification of the lantern slide is exactly the same as that of the original negative, whereas if a camera be used it is possible to reduce the image in such a way that the details of a large plate can be produced within the limits of a slide, or, if desired, we can magnify any portion of the field of the plate on to the lantern slide. It must be remembered that such processes are attended by complications which may spoil or damage the slide, whereas with the contact method we have reduced the procedure to its simplest elements. It is, in fact, no more difficult than gas-light printing. Those who desire to study a

simple text-book on the subject are recommended to read "Lantern-slide Making," by the Rev. F. C. Lambert, M.A., published by Hazell, Watson and Viney, Ltd. For the purpose we have in hand the contact-method satisfies all requirements.

(1) *Exposure.*—The photographer must select the source of light that he is going to use and work with that one throughout. Our experience has been with electric light, but it is possible to use oil lamps, gas, or magnesium wire. For the guidance of those who prefer to follow our example, it may be of interest to note that working with a six candle-power frosted lamp the exposure for an ordinary negative at 2 ft. was one of ten seconds when using the Ilford Special Lantern plate.

(2) *Preparing the Plate for Exposure.*—It is necessary to dust the negative carefully with a camel's hair brush, as otherwise particles of dust will cause what are spoken of as pin-holes in the slide. It is not, however, advisable to dust the magic lantern plate itself, for by so doing we develop frictional electricity, and so cause the attraction of dust particles. It is better to be content to give the edge of the plate a sharp rap on the table in order to shake off dust particles. In doing so we must be careful not to break the glass, as we have lately found some of the glasses to be very thin, possibly a result of "war economy."

The film side in lantern plates is not very easy to recognize, as the coating is thinner than that of ordinary negatives. If in doubt as to which is the film side, damp your finger slightly and try it on one corner of the plate, when the film will reveal itself by its stickiness.

We have found it of great advantage to take out four plates at a time, and to store those not actually in use in the black envelopes which can be purchased for the purpose at any photographic store. This avoids the trouble of opening, unpacking, and repacking the plates, first in their folds of paper and then in their original box.

(3) *Development of Lantern Slides.*—We have used hydroquinone as a developer, and have had every reason to be satisfied with it. It is an advantage to filter the solutions (both developing and fixing) in order to eliminate the presence of solid particles which might cause pin-pricks. One quantity of solution should not be used for more than four plates. The use of clean fixing solution for each batch of plates is imperative.

(4) *When to stop Development.*—There is no such thing as a perfect lantern slide. A density which is suitable for one light is quite unsuitable for another; the stronger the light the denser should the slide be made. When a strong slide is needed it is necessary to continue development until the high lights have begun to brown over as seen by reflected illumination.

(5) *When to stop Fixation.*—The danger lies in too short a time being allowed for this purpose, since the film, being thinner than that of ordinary negatives, clears with unusual rapidity. No harm will come from over-fixing if the bath be clean. A quarter of an hour may safely be allowed for the purpose.

(6) *Drying of Plates.*—It is important to dry the plates in a dust-free place, since every speck of dust is greatly magnified by the lantern. Not only that, but before the plate is placed in the drying rack it should be carefully wiped over with cotton-wool under water in order to remove any sediment without scratching the plate, and the glass side should be wiped dry with a cloth. Some people advocate drying under methylated spirit, but our experience in this respect has not been very fortunate. We prefer, if possible, to avoid unnecessary manipulation.

(7) *Reduction and Intensification of Lantern Slides.*—These processes should be carried out on the same lines as for ordinary negatives. The matter is dealt with at length in Lambert's book already recommended. It is a very distinct advantage to intensify or reduce certain negatives.

(8) *Varnishing of Lantern Slides* is not necessary, and entails the possibility of serious disadvantages arising unless the manipulation is very skilfully performed. The method of binding, masking, spotting and otherwise marking lantern slides is explained in Lambert's book.

(*November* 1, 1916.)

Bilateral Glioma of the Retina with Multiple Metastases.

By Frank E. Taylor, M.D., F.R.C.S., and Norman B. B. Fleming, M.B., Ch.B., Lieut. R.A.M.C.

A critical examination of the collected statistics of Wintersteiner [7] on glioma of the retina reveals the fact that when malignant characters are borne by this neoplasm they are shown by the extension and spread of the new growth in two ways, namely:—

(1) By involvement of the extra-orbital structures of the face and head, either (*a*) by direct continuity of growth into the bones and other structures adjacent to the orbital cavity; or (*b*) by direct extension of the growth backward into and along the optic nerve to the meninges

and the brain; or (*c*) by the occurrence of growths in the lymphatic glands in the neighbourhood of the parotid gland, and also occasionally involving that organ.

(2) By the formation of metastatic growths in various organs in distant parts of the body.

That the former condition is much more frequently observed than the latter is also shown by Wintersteiner's figures. Out of a total of 497 cases of glioma of the retina, regional invasion occurred in the bones of the cranium and face in forty cases, in the brain and meninges in forty-three cases, in the adjacent lymph glands in forty-six cases, in the parotid gland in nine cases, in the spinal cord and membranes in three cases; whereas metastases in distant organs were found in the skeletal bones in nine cases, in the liver in seven cases, in the ovary in two cases, in the kidney in two cases, in the lungs in one case, in the spleen in one case.

Further, in addition to our own case here recorded, in which metastases occurred in the brain and meninges, in the vertebræ, in the mesenteric glands, and in the ovary, we are able to find only five cases recorded since those collected by Wintersteiner in which metastases in distant organs were found.

In Fehr's [2] case the metastatic growths occurred in the ribs, sternum, and pleura, in the inguinal and mediastinal lymph glands, and nodules were also found between the left lung and pericardium, and adherent in the anterior mediastinum.

In Radcliffe and Goldberg's [6] case metastases were found in the liver, and in Lieber's [4] case in the lungs.

Dahs's [1] case is buried in the obscurity of a Greifswald *thesis* to which we are unable to gain access: we are therefore unable to state the distribution of the metastatic growths.

In Gardner's [3] case the organs affected were the sternum, ribs, costo-chondral cartilages, vertebræ, the pelvic bones, both femora, and both testicles.

With regard to the frequency of bilateral growths, the collected statistics of Owen [5] show the condition to be bilateral in rather less than 25 per cent. of the cases, for out of a total of 552 cases 417 were unilateral and 135 were bilateral.

Four facts connected with the following case are of sufficient interest to make it worthy of being placed on record, namely:—

(1) The presence of advanced growths in both eyes at the same time.

(2) The very advanced condition of the growths when the patient was first brought up for treatment.

(3) The presence of metastatic growths, both by direct extension into the brain and meninges, and by metastases in the vertebræ, mesenteric glands and ovary.

(4) The relatively small amount of shock and other disturbance which followed so severe an operation as complete evacuation of both orbits in so young a patient.

The history of our case is as follows:—

L. D., aged 3, was sent to the Royal Westminster Ophthalmic Hospital on February 14, 1916, and was seen by Mr. Elmore Brewerton, who ordered her immediate admission into hospital. Something

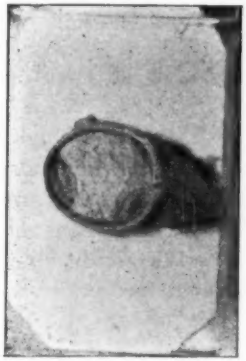


FIG. 1.

Hemisection of right eye.

FIG. 2.

Hemisection of left eye.

abnormal in the left eye was first noticed eighteen months previously, probably in September, 1914. No advice was sought till September, 1915, when she was taken to the hospital at Guildford. The mother states that she was then refused treatment, on the ground that it was useless. The child had not been fretful, was fairly well nourished, and ate and slept well.

On admission, a fungating growth protruded between the lids of the left eye, keeping them separate; it had been fungating for three months; the mass was moist with a thin semi-purulent discharge. The right eye was much enlarged and apparently proptosed. The iris was fully dilated and stretched into a thin band at the limbus. The lens was in contact

with the posterior aspect of the cornea, being pushed forward by a growth behind. This growth was of a lemon-yellow colour, as seen by ordinary daylight, and also by oblique focal illumination. On ophthalmoscopic examination no reflex was obtained.

As regards treatment, an operation was performed within an hour of admission. The left orbit was exenterated. The lids were partly removed and the wound stitched up, an aperture being left for packing. The right eye was excised. The conjunctiva was removed and the optic nerve was divided $\frac{3}{4}$ in. behind the globe. The nerve was found to be involved, being surrounded by growth for $\frac{1}{2}$ in. behind the eye. The right socket healed readily. The left socket became septic, and the discharge had an unpleasant odour, but rapidly improved when irrigated with hydrogen peroxide and dressed with iodoform.

On March 11 the child was discharged, there being as yet no sign of recurrence in either socket. While she was in hospital there was some bronchitis, from which she recovered, and at all times she took food with avidity.

The patient was re-admitted on March 30 with a large recurrence, equal in size to half a tennis ball, protruding from the right orbit. The left orbit was completely covered with skin, but, with a little pressure, a solid mass could be felt in the socket. The same day the right orbit was thoroughly cleared out, the greater part of the lids being removed at the same time. By this time the child was thinner, and was inclined to be fretful. She took the anæsthetic very badly and stopped breathing, but artificial respiration was successfully carried out. A general anæsthetic was required subsequently for the first two dressings, and the same difficulty with respiration was experienced. After this the child's condition improved, but a muco-purulent nasal catarrh developed, which persisted until death supervened about eight weeks later. The discharge was incessant, and before the end caused much excoriation of the face. The head was noticed to be disproportionately large, and emaciation was very pronounced.

The right socket quickly filled up again and the growth in the left one infiltrated the skin and became fungating, and both orbits were again cleared out on May 1. The greater part of the growths was found to consist of a material which had undergone pseudo-myxomatous degeneration.

After the final operation, the child quickly went downhill. She became quite apathetic, and neither ate nor slept. On May 3 she was found to have pneumonia of the left lung, but there were no rigors and

no rise in temperature or pulse. She cried a good deal, and her breathing was short and jerky, but there was little to indicate that she was suffering much pain. On May 6 the right lung showed signs of consolidation at the base, and death occurred on the evening of May 7.

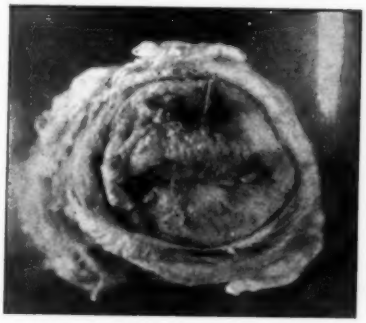


FIG. 3.

Recurrence from right socket.

PATHOLOGICAL REPORT.

The tissues removed from the orbits presented the following characters: Externally the right eye appeared normal except for a mass of new growth on the posterior aspect of the globe, embedded in which was the excised portion of the optic nerve. This mass was triangular in shape, being ½ in. wide and ½ in. from before backwards. On section, the coats of the eye appeared normal, but the anterior chamber was very shallow, the lens being pushed forward by a greyish-white growth which completely filled the vitreous chamber.

The left eye was enlarged and filled by a soft mass of new growth, which was everywhere confined within the coats of the eye, except in front where the lens and structures in front of it were missing, and the growth projected forwards between the lids, the surface being ulcerated and excavated. The posterior half of the neoplasm was of greyish-white colour, whilst the anterior half was of a dark brownish-red colour, as if infiltrated with blood. The structures of the eye, except the sclera, were entirely unrecognizable.

Microscopically, the growths were composed of closely packed small round cells, with large deeply staining nuclei, and extremely little

surrounding cytoplasm. A few spindle cells were also present. Small syncytial masses, with multiple nuclei forming the so-called giant-cells, were scattered irregularly throughout the mass in small numbers. The intercellular stroma was extremely small in amount, and was almost unrecognizable. There was much hæmorrhagic extravasation into the growth, although blood-vessels were scanty in numbers. Neuro-epithelial rosettes were not to be found in the sections.

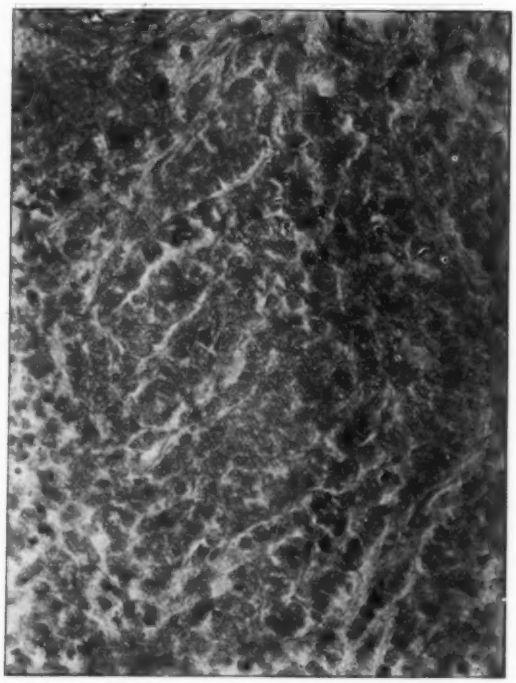


FIG. 4.

Section of glioma of eye showing masses of more or less degenerated round cells (feebly staining). (× 320.)

The choroid and optic nerve in both eyes were infiltrated with growth, so also was the left upper eyelid. In the right eye the structures anterior to the posteror capsule of the lens were quite free from growth.

POST-MORTEM EXAMINATION REPORT.

The body was that of an exceedingly emaciated female child, showing an extensive impetiginous rash on the face; both orbits, having been recently exenterated, showed gaping hollows revealing the base of the sphenoid.

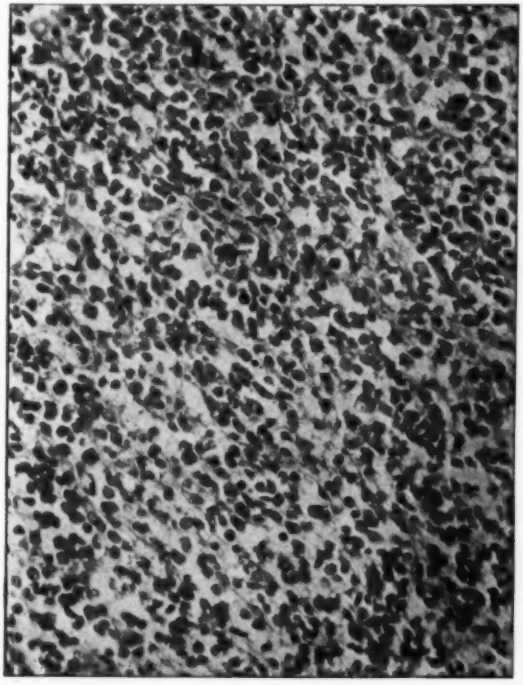


FIG. 5.

Section of secondary tumour of ovary, showing structure of round-celled sarcoma. (× 320.)

Removal of the calvarium was difficult, owing to the bone being adherent to the dura mater. This was found to be due to about twenty curious looking growths, round in outline, some of them being discrete and some coalescing. They were about $\frac{1}{4}$ in. in thickness, bright red in colour, like healthy granulations, and they varied in size from $\frac{1}{4}$ in. to

2 in. in diameter. The pia mater was hyperæmic in those areas, but apart from this the convexity showed nothing abnormal. The brain weighed 2 lb. 8 oz. At the base of the brain, in the position of the optic chiasma, and to some extent obscuring or replacing it, there was a large mass of growth covered by pia mater.

Both lungs showed a condition of septic pneumonia. The mesenteric glands were enlarged and were the seat of metastases. In the centre of the right ovary there was a blood-clot, measuring $\frac{5}{16}$ in. in diameter. This was surrounded by an area of new tissue, forming a zone $\frac{3}{16}$ in. in thickness. The right Fallopian tube was normal, so also were the left ovary and tube and the uterus and vagina. An oval mass of growth $\frac{3}{4}$ in. by $\frac{1}{2}$ in. protruded from the anterior aspect of the body of the second lumbar vertebra, and projected about $\frac{3}{8}$ in. above the surface of the bone.

The secondary growths in the meninges and in the ovary were examined microscopically and presented the characters of a small round-celled sarcoma, no neuroglial elements being recognizable.

Our thanks are due to Mr. Elmore Brewerton for his kind permission to publish this case.

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(*November* 1, 1916.)

Case of Recurrent Glioma of the Retina.

By Arnold Lawson, F.R.C.S.

The salient points of this case are as follows: The left eye was removed at Moorfields Hospital for glioma, the child, a boy, being then aged 3½. The condition of the eye after removal is described as follows by Mr. Greeves, who at that time was pathologist to Moorfields Hospital: "I find that there was a large scleral staphyloma in front, and that the thinned sclera was infiltrated with glioma cells—also that masses of growth could be traced along the sheaths of the vortex veins. The cut end of the nerve was apparently free of growth, although the proximal part (towards the eye) was infiltrated."

The boy remained well for a few months, when a recurrence of the growth in the orbital cellular tissue was noted. An exenteration of the orbital contents was then performed, but a few weeks later a fresh recurrence was noted and the child was sent to the Middlesex Hospital for treatment by radium. A large dose of radium was inserted into the growth and left there for four hours and a half. Free sloughing followed and on this account the child remained under in-patient treatment for about six weeks. At the end of this time all semblance of growth had disappeared and he was in excellent health.

Three months later he was re-admitted to hospital with a soft fluctuating swelling in the orbit, which was opened, and a considerable quantity of pus and pultaceous material evacuated. The condition was relieved for a time, but as the purulent discharge continued, an examination of the pus was made and *Staphylococcus aureus* was found in abundance. A vaccine was prepared and used but had no satisfactory effect. Further operative measures were tried about a fortnight later, and an attempt to clear out the cavity with the idea of using radium again. The swelling had increased a good deal by this time and very free hæmorrhage attended the operation and it was also found that the orbital bones were much eroded and that the antrum was open and full of grumous growth. Very severe recurrent hæmorrhage followed and lasted for two or three days, and further examination of the discharge led to the discovery of a streptococcus.

From this date the local condition went rapidly from bad to worse and the mass grew with extraordinary rapidity. The tumour spread over the parotid region, down into the neck, forwards through the skin, and inwards and backwards into the mouth. In spite of all, the child's general condition of health remained excellent till about three weeks before death, when rapid emaciation set in. Up to that time he was lively and cheerful and always ready to play with toys, and laughing and singing like any other happy child. Death occurred about three months after admission into hospital with the second recurrence.

Case of recurrent glioma retinæ. Photograph taken a fortnight before death. The superficies of the tumour is largely gangrenous.

The accompanying photograph, which was taken a fortnight before death, gives a good idea of the extraordinary rapidity of the growth and its enormous size. It is to be remembered that the period between admission into hospital and death was only three months, and that at the time of admission the growth was no bigger than a tangerine orange and then consisted chiefly of pent-up pus. The measurements of the growth, taken after death, were as follows: Circumference, 16¾ in.; vertical diameter, 12½ in. The weight was 43 oz.

The following précis of the post-mortem notes made by Dr. Cockayne are of interest: "The bones of the face were eroded and the growth

had pushed its way into the mouth, displacing the jaws with their teeth. Inside the skull the tumour had penetrated through the sphenoidal fissure, and there was a large subdural mass which had flattened the under surface of the temporal lobe and had pushed back the anterior portion of the temporo-sphenoidal lobe. The olfactory lobe on the same side was flattened. Both optic nerves were quite free of growth so far as could be seen, and this was subsequently confirmed by microscopical examination. (The slide showing sections cut from both nerves is on view.) The growth had crossed the middle line under the dura mater in front of the optic nerves and chiasma—i.e., it extensively invaded the anterior fossa of the skull on both sides. The brain showed no abnormality whatever beyond displacement. All the organs of the body were perfectly healthy and there was no glandular involvement anywhere."

Microscopically the tumour in section (of which a slide is shown) is extremely cellular, with extremely slight supporting connective tissue. The cells are small and round or oval in shape, with large nuclei, and practically no protoplasm.

The outstanding features of the cases are, I think, the following :—

(1) The failure of radium treatment.

(2) The purely local nature of the recurrence, which was practically a continuation of the original growth outside the globe.

(3) The extremely rapid growth of the tumour when once it had been opened up, and after attempts had been made to clear it away.

(4) The total absence of true metastases and the complete freedom from involvement of structures such as the cerebrum, which, though covered by the meninges, were otherwise in immediate contact with the growth.

(5) The excellent general condition of the patient, whose health was not materially affected until the protrusion of the growth into the mouth set up acute septic absorption from which the child died.

The most interesting point is the question of metastases. Of course it is now well recognized that retinal gliomata or glio-sarcomata, as I think they should be called, are not very prone to give rise to metastatic deposits, except purely local recurrences in the immediate neighbourhood; but certainly such do occur sometimes, and it is strange that this extremely malignant growth exhibited absolutely nothing of the sort, not even implicating the stump of the optic nerve, which was lying all the time embedded in it.

The failure of radium treatment was unfortunate, because it

appeared to be an excellent case for its use, and it really seemed at first as if it had effected a cure.

That the patient died directly from septicæmia there can be no doubt, and, as I have pointed out already, a most striking feature was the indifference of the child to this horrible growth, to which, until the mouth was invaded, it paid no attention except when dressing-time came round.

The growth itself caused the child no pain at all, and I never had to give any morphia or other anodyne. The worst part of the case towards the end was that the growth became gangrenous in places and the fœtor was intense. A free use of H_2O_2, and frequently applied wet dressings soaked in weak Sanitas solution, answered the best of the many things tried with a view to mitigating the fœtor.

DISCUSSION.

Mr. Wm. C. Rockliffe (Hull): Mr. Arnold Lawson's, Dr. Taylor's and Mr. Fleming's papers remind me of three similar cases of recurrent glioma occurring in my own practice. In the first case, the patient, aged 3, was brought back six weeks after the excision with a rapidly increasing recurrent growth in the orbit about the size of a walnut, which had been noticed about fourteen days previously. Six days later the growth had more than doubled its size, and an exenteration was done, the child dying shortly afterwards with glioma of the brain. Owing to the child taking the anæsthetic badly the primary excision was hurried and the nerve was divided closer to the eyeball than was desirable. Mr. Parsons reported: "The recurrent growth is not metastatic, but has invaded the whole length of the nerve by direct continuity, and has entirely replaced the nerve fibres. It also invades the other orbital tissues widely, and throughout has the typical appearance of a glioma of the retina." In the second case, a child aged $3\frac{1}{2}$, the primary gliomatous growth extended deeply into the orbit and the excision was complicated by very free hæmorrhage, and again the nerve was divided nearer the globe than was intended. The patient was re-admitted, twenty-four days later, with a large recurrent growth in the orbit protruding $\frac{3}{4}$ in. in front of the superciliary margin. Exenteration was done at once and the late Mr. Coats reported "typical glioma." A week later a *second recurrent* growth commenced rapidly filling the orbit, and as the child showed cerebral symptoms the patient was removed by its parents. Four days afterwards *extraocular metastases* developed over the occipital bone and steadily increased in size, and the child died about two months after the primary excision, extremely emaciated with secondry growths on the skull and lower jaw. This case, even to actual dates, is almost identical with Mr. Fleming's, and, like his and my first case, succumbed in two months after date of excision. In the third case, a child aged 3, the recurrent growth

did not appear until six months after excision of the left eye. It was accompanied by cerebral symptoms. A month later the child was in a comatose condition, with twitching of the right arm and face, and apparently right hemiplegia; there was also considerable ecchymosis of the right orbit. The child died in convulsions six weeks later—i.e., nine months after the excision. Mr. Treacher Collins reported it as a case of "typical glioma."

Mr. E. TREACHER COLLINS: With reference to that last remark made by Mr. Rockliffe, I should like to say that I do not regard glioma of the retina as a sarcoma. It is best spoken of as neuro-epithelioma. It is interesting to contrast the way in which different forms of malignant intra-ocular growths spread, that is to say, glioma as compared with sarcoma, the one beginning in the neural epiblast, and the other originating in mesoblastic tissue. Sarcoma of the choroid generally spreads by metastasis: the large majority of the patients who die from sarcoma of the choroid die from metastatic growths. But the gliomatous growths, as we have heard to-night, and as I found in a series of cases traced out in association with Mr. Lawford, usually spread by continuity. Recurrences when they occur are usually local, in the orbit, about the face, or in the brain. In trying to find an explanation of the difference in the way in which these growths spread, we may, I think, look to the work of Mr. Sampson Handley, who showed that cancer of the breast usually spreads by "centrifugal lymphatic permeation." This is probably also the case in glioma of the retina, while sarcoma of the choroid is spread by emboli of sarcoma cells carried in the blood stream. As a clinical point, I would like to ask members of the Section whether they have ever checked the spread of a gliomatous growth by doing an exenteration of the orbit. I have done exenteration many times, but have never, in these cases, saved the patient's life.

Mr. HOLMES SPICER: In 1901 I had a case of glioma of both orbits in a child. Mr. Brewerton helped me to remove the left eye, the other had been previously removed for glioma: a long piece of the nerve was taken; the eye was full of glioma, but the cut end, examined by Mr. Brewerton, was healthy. Two months later there was a recurrence in the left orbit about as big as a filbert; the orbit was evacuated; the removed part was examined by Mr. Brewerton, and was reported on as gliomatous, but unfortunately the note of the detailed examination cannot be found. The patient is alive now after fifteen years.

Mr. ELMORE BREWERTON: I may say I exenterated the orbits of this child four or five times, but I did not do so with the idea that I was thereby saving the patient's life, but rather to save some of the trouble to the nurses at the hospital in looking after the patient. The cleared-out orbit was far easier to dress than was a fungating mass, which, as Mr. Lawson has already said, smells horribly, although it is not painful to the patient; and I am sure the patient is more comfortable after exenteration. If the growth is limited to the orbit I think exenteration should always be done. With regard to the case mentioned

by Mr. Holmes Spicer, the eye condition was not quite a typical glioma: it was at a late stage, and showed no rosettes. The nodule which was subsequently removed from the apex of the orbit showed the usual round cells of recurrent glioma.

Mr. J. H. PARSONS: I should like to protest against the inclusion of glioma among the sarcomata.

Lieut.-Colonel ELLIOT: I agree with Mr. Brewerton in what he has said. In India I saw many of these cases, in the condition of masses fungating from the orbit. When the growths were confined to the orbit, there was no question that an enormous difference was made to the patients' comfort—especially if they could not be kept indefinitely in hospital—by the performance of exenteration. Sometimes I had to do it more than once, but do not remember a single case of such a patient dying on the table or in hospital after operation. Although I knew I was fighting a losing battle, the object was to confer increased comfort on the sufferers.

Section of Ophthalmology.

President—Mr. William Lang, F.R.C.S.

(*February* 7, 1917.)

Rupture of the Optic Nerve (?).

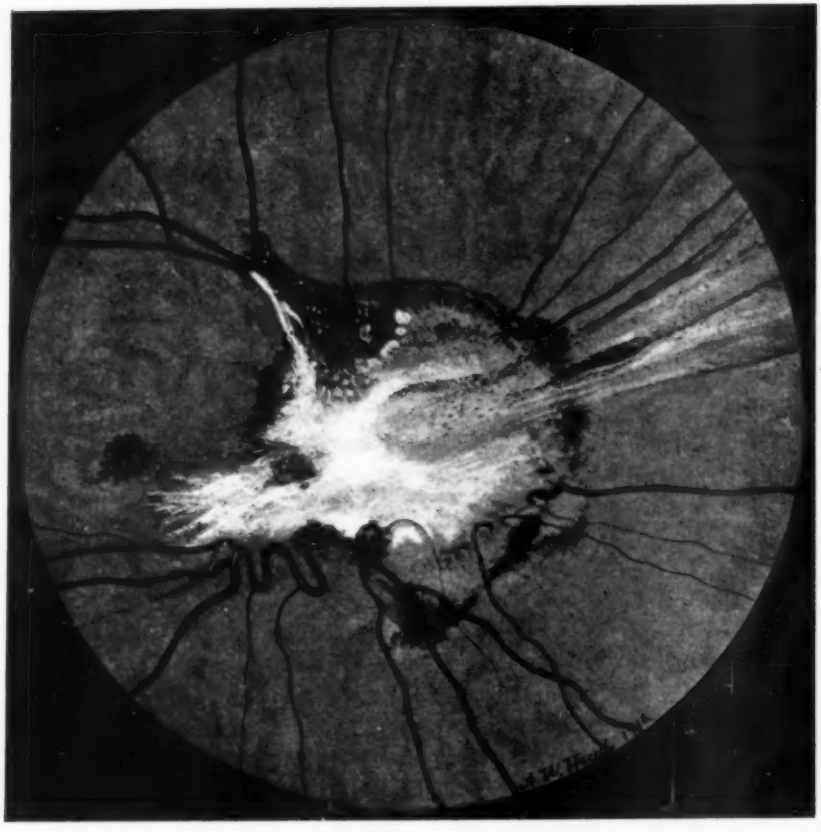
By Gustavus Hartridge, F.R.C.S.

Lieutenant D., of the Royal Flying Corps. In August, 1916, he was flying in France and when about 3,000 ft. up, he suddenly fainted and remembers nothing more until five days later when he regained consciousness and found himself in hospital. An observer who saw the accident said that the machine came down gradually at first, but the last 500 yds. it fell vertically and was smashed to pieces. The patient sustained serious damage to the left arm and the right eye, as well as many small injuries; no apparent skull injury.

When I saw the patient on September 22, three weeks after the accident, there were extensive subconjunctival hæmorrhages in both eyes. Right eye: Pupil widely dilated and inactive to light; the vitreous was full of blood and no details of the fundus could be seen; there was a small punctured wound through the upper lid, immediately below the supra-orbital notch; a probe passed into the puncture only went a small distance into the orbit and no foreign body could be felt. The vitreous hæmorrhage gradually became absorbed, and the present condition of the fundus is well shown by the drawing (*see* next page). Left eye: Vision $\frac{6}{5}$, fundus normal.

The President: I remember seeing a boy who had been poked in the eye by a clothes-prop, and the lower portion of the optic nerve was torn away from

the margin of the disk. At first, one could not distinguish anything except a large hæmorrhage and a deep excavation. The case is recorded in the *Transactions* of the Ophthalmological Society.[1] Subsequently there was only a half-field, the absent upper half corresponding to the torn lower half of the disk. It appeared, ophthalmoscopically, very similar to what one sees in coloboma of the optic nerve sheath.



Rupture of the optic nerve (?).

[1] *Trans. Ophth. Soc. U. K.*, 1901, xxi, p. 98.

(*February* 7, 1917.)

Unusual Condition of Choroid (? Congenital).

By HAROLD GRIMSDALE, F.R.C.S.

M. B., FEMALE, aged 23, attended St. George's Hospital as a medical out-patient. The condition was diagnosed as hypothyroidism and she was looked upon as a sporadic cretin. As her vision was obviously defective, she was referred to the ophthalmic department, when the appearances were noted. In both eyes there is an area of choroid tissue, of rather darker colour than normal, surrounding and including the disk and macula; the shape is subovate with the long diameter, which is roughly twice the vertical, transverse. The remainder of the fundus shows the white colour of the sclera, crossed by the retinal vessels and a few large choroidal trunks. There are a number of patches of pigment, not heaped-up but flat, scattered over the whole. Her vision is $\bar{c} - 5$ sph. $-$ 3 cyl., $\frac{6}{6}$ in each eye, and binocularly $\frac{6}{36}$ doubtfully. She is stupid, and the field could not be mapped out but it is clearly much contracted. A Wassermann reaction has not yet been obtained.

I take it this is a congenital absence of choroid, but it differs from the only other case of the kind I have seen, partly because the area of choroid left is much more irregular in shape, but more so because of the pigment in the rest of the fundus. I have never seen anything like these large irregular masses of pigment, not heaped up, but apparently lying flat over the vessels, and scattered over the greater part of the white sclerotic.

(*February* 7, 1917.)

Three Cases of Fur Infection of the Conjunctiva from Cats.

By ARNOLD LAWSON, F.R.C.S.

ALL three cases occurred in children whose parents were of the prosperous classes, and whose homes were therefore of the cleanest and best. I have never personally come across a similar case occurring in an adult.

Case I.

My notes on this case, which occurred some years ago, are not satisfactory as I have somehow lost that part of them which referred to the pathological examinations. The patient was a girl, aged 15, an only child, who somewhat lacked playmates and was devoted to a Persian cat. One evening shortly after having had an extra romp with the cat during which she had frequently buried her face in the cat's fur in the way children are so fond of doing, her eyes became suffused and she also developed a bad sneezing fit. The next day her eyes and nose were swollen and discharge began to appear, and by the evening, when I first saw the girl, the affection was acute. Both eyes were streaming with yellow purulent discharge and the lids were much chemosed. The nostrils were similarly greatly swollen and purulent; slightly sanguineous matter was freely oozing from them.

Cultures were taken of the discharge and also from the cat's fur. My recollection of the result, which as stated I have unfortunately lost from my case book, is that the *Staphylococcus pyogenes aureus* was in abundance in the eye discharge, and that the same organism was isolated from the fur.

Energetic treatment on conventional lines soon effected improvement, and the child eventually made an excellent recovery without any damage to the eyes.

It was for two or three days a case of great anxiety, as it was quite one of the most acute cases of purulent conjunctivitis that I have ever seen. One has, of course, to be careful in such cases before definitely assigning the cause, but here there seemed no other possible explanation as the girl was living in the best of circumstances and was perfectly well until the cat episode occurred. I need scarcely add that the cat met with an untimely end.

Case II.

This case also occurred in a child of well-to-do people. The patient was a boy aged 14, and his case was extremely complicated and severe. The history was as follows: About a fortnight before I first saw the patient, a slight inflammation was noticed in the left eye, accompanied by slight discharge and a slight swelling over the left parotid. The local doctor thought that it was a case of mumps and the boy was isolated. The swelling however did not run the course of mumps, and

as it gradually grew larger and the conjunctivitis increased in spite of zinc lotion, he was sent up to me. During this fortnight there was no fever and no pain.

When I saw him I noticed that the whole left side of the face was much swollen and that over the parotid there was a brawny induration not particularly tender, but with the skin slightly reddened over the angle of the jaw. Extending into the neck and almost continuous with the parotid swelling was another indurated mass evidently consisting of swollen glands. The lids of the left eye were considerably swollen and eversion for examination was difficult. When this was effected the conjunctival surfaces of both tarsi were seen to be fairly covered by coarse granulations especially marked in the upper lid, where, near the fornix, there were some bunches of exuberant granulation tissue. The retrotarsal portions of both sacs were much chemosed with great enlargement of the follicles, and a fairly abundant muco-purulent discharge lay in the furrows of the swollen tissues. The bulbar conjunctiva was also considerably swollen and rather pink in colour. The cornea and all other structures were quite healthy.

The cause and nature of the trouble seemed at first obscure. Naturally, suspicion of tubercle crossed my mind, but I dismissed it as improbable on account of the rapidity with which the disease had developed. There was, further, a history of a Persian cat of which the boy was very fond, and he was in the habit of walking about with it perched on his left shoulder; he cuddled it at irregular intervals. Systematic investigation was the only possible way of arriving at the causation, and in the meantime the boy was put under an anæsthetic and the disease treated by thorough cutting and scraping, finishing up by thoroughly swabbing out the upper and lower sacs with a 2 per cent. solution of silver nitrate. The boy did very well and the glandular swellings at once began to subside. However, steady treatment for about six weeks with argyrol, weak copper drops and an occasional touch of mitigated silver stick to spots where the granulations threatened to recur, was necessary before he became really well, and the glands were not completely normal again for a little while longer still.

Now as to the investigation. The scrapings were put into sterile tubes and submitted immediately after operation to expert examination, and from them were isolated (*a*) *Streptococcus longus*, (*b*) *Streptococcus brevis* and (*c*) *Staphylococcus albus*. Obviously the streptococcus was the important organism and examination was directed to seeing if this

could be found in the cat. The question arose as to whether any other animal but the cat was likely to be a factor, and as there was also a pet dog in the house (of which however the boy was not particularly fond) it was decided to examine both.

Portions of the hair and swabbings of the skin were taken in each case. The dog's hair revealed (*a*) *Staphylococcus albus*, (*b*) *Bacillus subtilis* and (*c*) *Proteus vulgaris*, whilst from the skin swabbing were obtained (1) *Staphylococcus aureus* and *albus*, and (2) *Micrococcus catarrhalis*. In the cat the chief organism found in the hair was a *Staphylococcus albus*, whilst in the skin swabbing a number of organisms were found, the most important being (*a*) *Streptococcus brevis* and (*b*) *Staphylococcus citreus*, the other organisms consisting largely of *Bacillus proteus vulgaris* and the *Bacillus subtilis*.

It was therefore only in the case of the cat that an organism, a streptococcus similar to that found in the lid scrapings was discovered. This fact, coupled with the history already given of the boy's fondness for the cat gave the verdict unhesitatingly against the cat as the causative agent. The dog being found "not guilty" was acquitted, whilst the cat was summarily executed.

Case III.

This case was unquestionably the worst of the three: for if the trouble had not been eradicated it might undoubtedly have led to the child's death. It occurred in a little girl aged 5. When staying with some friends of her parents at a place in the country to which there was a farm adjacent, she contracted a muco-purulent conjunctivitis of a mild type. The history of its onset was that it supervened very shortly after nursing a cat which was a great favourite with her and which divided its favours impartially between the child and the denizens of the neighbouring cowsheds. The conjunctivitis proved intractable to ordinary treatment and the child was taken to an expert for advice. Still treatment did not improve matters and after a time the submaxillary and sternomastoid glands on the same side began to enlarge. And so the case drifted on for two or three months, the eye apparently remaining in much the same condition of chronic mild conjunctivitis, and the neck trouble ever growing more formidable. Ultimately I was called in to see the child and on everting the upper lid I found a mass of exuberant cock's-comb granulations occupying the greater part of the retrotarsal tissue and trespassing on the posterior edge of the tarsal

plate. The lower sac was perfectly healthy except for a little reddening and the lesion must have been discovered had anybody taken the trouble to evert the upper lid.

The clinical appearance combined with the history and enlargement of the cervical glands made me at once suspect that it was a case of conjunctival tubercle.

I at once took the child into a nursing home and under an anæsthetic cut and scraped away all the infected parts, finally with due precautions coating the whole operated surface with mitigated nitrate of silver stick. A certain amount of reaction followed as a matter of course, but warm boracic bathings and pads, with the free use of weak iodoform ointment answered very admirably, and the child made an excellent and complete recovery. I warned the parents that if the glands in the neck were tubercular and their condition resultant on the conjunctival growth, the scraping and cutting away of the latter would probably be followed by an outburst of inflammation in the glands, and this is precisely what happened. Three little operations in the course of the next few months had to be performed on glands which, in one case at any rate, broke down, but ultimately the neck cleared up thoroughly well, and now four years after the lid operation, the child is quite well, without the slightest sign of recurrence either in the eye or glands.

The scrapings were submitted to Dr. Browning for examination. He reported that direct smears made from the ground-up scrapings showed the presence of a very few bacilli, which were very short and fat, and resembled the bovine type of tubercle bacilli. Some of the scrapings were injected into the subcutaneous tissue of a guinea-pig, which rapidly wasted and died in a fortnight. The guinea-pig after death showed a small area of necrosis round the inoculation site with slight enlargement of the corresponding inguinal glands, but no other lesion. Microscopical examination of sections from the local lesion and from the glands showed abundant tubercle bacilli. Dr. Browning adds that " these tubercle bacilli were in his opinion of the bovine type and the rapid death of the guinea-pig from the inoculation of extremely few bacilli (only five were found in twelve slides of the scrapings after prolonged search) tends to prove their bovine origin."

The guilt of the cat could unfortunately only be fixed by circumstantial evidence, because in spite of my best endeavours I could never succeed in getting hold of it. The owners, indignant at the imputation cast upon their pet, and solicitous for its welfare, stoutly and perhaps

advisedly, refused to allow even an examination of its fur and skin. Still I think the very great probability that the child was infected with the bovine type of tuberculosis, and the fact that the cat was a farm-yard cat, and was a pet of the child who was continually hugging and cuddling it, renders the ætiology of the case pretty clear and makes it fair to assume that the cat was the means by which the child was infected.

I have drawn the attention of the Section to these cases, not because there is anything specially new or interesting in them, for a fair number of similar fur infections of the eye already have been reported, but chiefly because I do not think the matter has been properly brought before the public, who do not sufficiently realize the danger of cats as a means of disseminating disease. The germ-carrying guilt of rats, mice and flies and other noxious creatures, which are in themselves a nuisance and are generally hated, has been cordially recognized, and means to deal with them by extermination are eagerly welcomed, but although I believe the cat is as bad an enemy in this way as any animal or insect, I fear that any attempt to exterminate these wandering nocturnal curses would meet with a hopeless opposition. I think the public, as a body, still believes in the fallacy of the cat being more cleanly than most other species of animals and still quotes the argument of the free use of its tongue and saliva on its fur to clinch the subject; and so I fancy the only thing to do is to impress more forcibly the fact that the cat as a pet is a source of danger, chiefly to children, who naturally love to cuddle it and bury their heads and faces in its septic fur.

(*February* 7, 1917.)

The Retinal Signs of Arteriosclerosis compared with those due simply to Increased Blood-pressure.

By P. C. Bardsley, M.B.

In 1897, in vol. xviii of the *Transactions* of the Ophthalmological Society of the United Kingdom, Mr. Marcus Gunn published his monumental work on the retinal signs of arterial sclerosis. From the clinical side there has been little or no advance since. Mr. Gunn laid down four cardinal signs of sclerosis of retinal vessels, viz.:—

(1) A tendency to *tortuosity of the arteries*, especially the smaller vessels.

(2) *Variation in the calibre* of the vessels, especially sudden diminution for a short length passing again into normal size. The calibre is apparently sometimes narrowed by nodules in the vessel walls, imparting to the vessels a beaded appearance.

(3) *Alteration of the normal light streak.* This becomes very much brighter and very much more sharply defined.

(4) *Indentation of veins*, either by supra-pressure of the artery crossing above the vein, or infra-pressure from crossing below. When there is considerable indentation there is great obstruction followed by all the sequelæ of back pressure, viz., stasis, thrombosis, œdema, and hæmorrhages into the surrounding tissues.

It is necessary for the clear understanding of this paper to keep these four signs all the time before us: tortuosity; variation of calibre; alteration of light streak; indentation of veins.

These signs of Marcus Gunn's have been confirmed by the ophthalmoscopic examinations of hundreds of observers since; but no additional sign of any importance has been added. De Schweinitz emphasizes the brick-red discoloration of the disk as a late but important sign; this is due to the dilatation of the capillaries and is the consequence of the venous obstruction and the back pressure.

Mr. Gunn was undoubtedly one of the first who advanced the opinion that arteriosclerosis was not confined to old age, and that old age was not alone productive of these changes. The prognosis of these cases was at first regarded as very bad indeed. Some years back, however, I came to the conclusion that this dire prognosis did not apply nearly so universally as was at first supposed. In following up the after-history of these patients I found that some with very advanced mischief, such as considerable retinal œdema with hæmorrhages, improved marvellously under treatment, and in some cases, to my surprise, the cardinal signs largely disappeared. At times, also, cases were published in the journals, such as the following, which appeared in the *British Medical Journal* of August 6, 1910. The case was published by Dr. W. M. Crofton, lecturer in special pathology, University College, Dublin, and was entitled: "Recovery from Apparent Arterial Sclerosis." This is the gist of Dr. Crofton's publication:—

Patient, aged 60, manager of a large commercial concern, had led a strenuous life. He was very short of breath, and suffered from

palpitation. A specialist diagnosed cardiosclerosis, and ordered him a sea voyage. His urine contained a trace of albumin, but no sugar. Owing to a feeling of weight on the top of his head and aching of the eyeballs, he was sent to Mr. Claud A. Worth. Mr. Worth's report was: "Retinal arteries are very small and tortuous. Central light streak very wide. Where an artery crosses a vein, the latter is so compressed as to be apparently interrupted. No retinal œdema. No hæmorrhages." Patient was placed under treatment for three months, after which he returned to work. Two months later he again saw Mr. Worth, who reported as follows: "Retinal arteries are large and have lost the 'silver wire' appearance. The appearance of interruption of the veins is much less marked; in fact, the arteries appear normal for a man of his years."

The publication of cases like this, and my own observations, compelled me to ask myself repeatedly this question: Can sclerosed vessels change in appearance in this manner? Are these cases which often show advanced signs (for surely back-pressure and œdema must pass as very advanced signs), are these advanced cases, which improve so much, incipient sclerosis, or are they sclerosis at all? We know that iodides may produce absorption of round-celled infiltration; and we may reasonably conceive of resolution of the hypertrophied muscle cells and elastic fibres; but are we justified in expecting the disappearance of organized granulation fibres and connective tissue? General experience answers—no. May not the signs, then, be due to some cause other than sclerosis?

Again, I observed that these changes could occur with *startling rapidity*. I saw patients, whose eyes I had carefully examined only a very short time before (in some cases only a few days), and whose vessels had appeared perfectly healthy and normal, and I saw those patients after a severe attack of influenza, scarlet fever, or some other toxæmia, develop these signs of retinal sclerosis, with deep indentation of veins, and a macular œdema sufficient to reduce their vision from normal to $\frac{6}{12}$. It was impossible to believe that an advanced fibrosis had occurred in a few days, nay, in a day or two. It was equally impossible to believe that these signs could be due to round-celled infiltration preliminary to fibrosis, for in many cases, after lasting for months, the signs largely or entirely disappeared. In these months the infiltration would surely have become organized.

I became certain, then, that these cardinal signs of Marcus Gunn did not *necessarily* indicate arterial sclerosis, but were often due to some accessory cause.

Many years ago at Moorfields, when observing these cases, I began taking the blood-pressures of the patients with a Riva-Rocci manometer. In nearly all the cases the blood-pressure was raised considerably above the normal, and in many cases excessively so. Often the patients sought advice for headaches which they thought were due to their eyes, but which were not relieved by suitable lenses. I commenced treating these patients with depressor drugs, strict diet, exercise, baths, &c., as recommended by the various authorities on blood-pressure. I noticed that when I was successful in lowering the blood-pressure to any considerable extent, along with the relief of the head symptoms, the ophthalmoscopic signs of retinal sclerosis appreciably diminished. Increased blood-pressure, then, accounted for some of the cardinal signs. When the toxic cause of the high blood-pressure was traced and was successfully removed by methods such as the complete clearing of the mouth of septic stumps, the improved changes in the retinal vessels were still more remarkable.

If, then, some of these cardinal signs were produced by the high pressure of the blood alone without sclerosis, while other signs were due to sclerosis, it became important to decide if it was possible to distinguish between the changes due to the simple raised pressure and those due to the more serious sclerosis. In the discussion on retinal vascular disease at the Ophthalmological Society, during the Session 1912-13, I ventured to state that I thought it was possible with the ophthalmoscope to distinguish between the signs of high tension and the signs of angiosclerosis.

The following seem to me the distinguishing characteristics. In simple high tension :—

(1) The vessels have an appearance of *uniform* distension and fullness.

(2) The light streak is *broadened* out: it may be greatly increased, apparently reaching almost the whole breadth of the vessel.

(3) The light streak is very much brighter than normal, the brilliancy increasing with the increase of the tension, until with very high tension it becomes like bright copper wire (not silver wire).

(4) The tight arteries indent the veins; with medium high tension they indent them slightly, with very high tension they indent them deeply, leading to back-pressure and all its consequences.

The following signs indicate sclerosis :—

(1) *Irregular* tortuosity, especially of the smaller twigs.

(2) Increased brilliancy of the light streak, while at the same time the light streak appears *narrower* and more central.

(3) Irregularity of calibre and beading are sure indications of sclerosis.

(4) General diminution in the size of the vessels and "silver wire" reflex show advanced sclerosis.

Now, of course, these two conditions of high tension and sclerosis very frequently coincide, but not by any means always. I have seen cases of definite early sclerosis of the retinal vessels with only slightly raised blood-pressure. The blood-pressure has been suddenly raised by some toxic infection, or by artificial means, and the above signs of high tension in the retinal vessels have been added to the previous signs. These signs have again disappeared on lowering the blood-pressure or eliminating the toxic cause. On the other hand, in advanced "silver wire" sclerosis, I have never seen the signs of sclerosis removed. I have seen some diminution in the intensity of reflex when the vascular pressure has been lowered by drugs, or the heart pressure has failed; the ophthalmoscopic signs of sclerosis always remained in evidence.

The question whether *continued* high blood-pressure induces sclerosis perhaps scarcely concerns us here. But, for my part, I do not think it necessarily does. For instance, there may be a raised pressure from aortic disease, with perfectly compensated hypertrophy. This may last for a lifetime without any signs of vascular sclerosis becoming evident. Again, in myxœdema the blood tension is raised (probably from the imbalance of the suprarenal and thyroid secretions) without signs, that is, retinal signs of sclerosis. When continued high tension is followed by sclerosis this sclerosis may be due to the same toxin which caused the high tension. Now, to confirm the above deductions, I called in the assistance of my House Surgeon, Mr. du Toit. We took a number of cases in the Salisbury General Infirmary, and artificially raised the blood-pressure. We selected, as far as possible, healthy cases from the surgical side of the Infirmary, such as males admitted for radical cures of hernia. We carefully examined the retinal vessels of each patient by a good illumination from a fixed light, and with a mydriatic in the eyes; and we made careful notes of the conditions of the vessels. We then took the blood-pressure with a manometer and noted it carefully from two or three examinations. We next administered a hypodermic injection of 5 to 8 minims of adrenalin chloride in normal saline, and the blood-pressure was recorded at intervals until it was found that there was no further rise. This was roughly one to one and a half hours after injection. Some cases responded well, the blood-pressure rising 40 to 45 mm.; others to a lesser extent, with a rise of 20 to 30 mm.; and in

some there was no rise of blood-pressure after the injection of adrenalin chloride. When the pressure was considerably raised, then another careful observation of the retinal vessels was made and a description of them accurately noted down.

As these experiments can easily be repeated by any ophthalmic surgeon, further details are not of any value.

From this artificial raising of the blood-pressure, we observed in the retina :—

(1) Increased fullness of the arteries.

(2) Increased brilliancy of the light streak.

(3) Increased breadth of the light streak.

(4) (*a*) Indentation of veins, where previously not indented ; (*b*) increased indentation of veins (in a few cases, very much increased), where previous slight indentation existed.

(5) The reduction of all these signs on the return of the blood-pressure to its former level.

From long experience, and from the above experiments, I think it is even possible to indicate roughly the actual height of the blood-pressure in millimetres of mercury from the observations of the retinal vessels alone. Where there are no definite signs of sclerosis, I think : (*a*) very slight indentation of veins, full arteries with broad light streak, indicate 145 to 150 mm. ; (*b*) very brilliant broad light streak with deeper indentation and slight signs of obstruction indicate 150 to 160 mm. ; (*c*) very brilliant broad light streak with apparent obliteration of veins indicate 160 to 180 mm. Where the signs of advanced sclerosis are present, the veins may be indented and nipped at lower tensions.

In conclusion, there are, I think, one or two pitfalls which it is necessary to avoid :—

(1) *The Pitfall of Faulty Examination.*—In a case of high arterial pressure, it is frequently a matter of great difficulty to ascertain whether thickening of the vessel wall is present or not by a *digital* examination of the superficial vessels. It is here that the ophthalmoscope plays so important a part. The vessels can be seen as they are in life, and by the direct method of examination the image becomes magnified about fourteen times. Hence the skilled ophthalmoscopist is able to detect change from the normal of a very slight amount, although to the inexperienced observer only very gross changes are detectable. Here lies the pitfall : Errors of refraction, especially high errors, blur the picture, and unless they are carefully corrected, the interpretation of what appears is fallacious.

(2) *The Pitfall of Acute Toxæmia.*—In patients suffering from very severe toxæmia, we *may* see in the retina: (*a*) Broad arteries; (*b*) increased light streak; (*c*) engorged veins; (*d*) some very slight indentation of these engorged veins. Yet the blood-pressure, as shown by the manometer, is abnormally low. Such a condition one may see, for instance, in an acute Bright's disease, a very severe influenza, &c. The explanation of the appearance of the retinal vessels is, I think, due to: (*a*) relaxation of the vasomotor control of the arteries; (*b*) dilatation of the heart, with loss of its muscular power; (*c*) consequent engorgement and stagnation of the venous system. This general condition is due to the toxin circulating in the blood. In several of these cases I have listened to the heart's action, and found the sounds so weak as to be almost inaudible. On administering small doses of digitalis, with an apparent general improvement in the patient, the blood-pressure has gone up with a jump, say, from 90 to 140 (this without any marked diminution of the albumin excreted in the albuminuric cases).

(3) *The Pitfall of the Failing Heart.*—A somewhat similar pitfall exists in cases of prolonged high tension with sclerosis, in which the heart's action suddenly begins to flag. In such a case the retinal signs would indicate to the careful observer a fall in the blood-pressure from what it previously had been, *yet* those signs would point to a higher pressure than the manometer would give. Why is this? Again because: (1) with the failing heart, the stagnation of the blood stream produces swollen veins; (2) owing to the hardness of their walls the arteries remain rigidly fixed; (3) the swollen veins *appear* to be obstructed by the rigid arteries. Keeping in mind these three liabilities to error—viz., faulty examination, acute toxæmia, the failing heart—I think it is possible for the ophthalmic surgeon to distinguish by means of the ophthalmoscope between increased blood-pressure and arteriosclerosis, and to base his prognosis and treatment thereon.

DISCUSSION.

Mr. HERBERT PARSONS: I do not wish to criticize the paper, but I should like to draw the attention of the author and the members generally to some work done by Mr. Foster Moore, which has just been published in the *Quarterly Journal of Medicine*,[1] and which I have had the opportunity of reading. That work bears directly on what Mr. Bardsley has told us, and it is an extremely

[1] *Quart. Journ. Med.*, Oxf., 1916-17, x, pp. 29-77.

important contribution to the subject. I mention it because it is published in a journal which is liable to be overlooked by ophthalmologists.

Mr. J. B. LAWFORD: This paper of Mr. Bardsley's is a very valuable contribution to the ophthalmoscopic evidence of vascular changes. I had the privilege of being a colleague of the late Mr. Marcus Gunn at the time he was observing the cases which he subsequently published, and I saw a great many of them. As probably every one will agree, Mr. Gunn was one of the best ophthalmoscopic observers we have ever had, and I consider that Mr. Bardsley has proved himself an apt pupil of his teacher. At the time, I accepted Mr. Gunn's statements fully, especially with regard to prognosis, in the class of cases he described. But not long afterwards I had it brought to my knowledge that the prognosis was not so grave, at all events, in all cases as Mr. Gunn led us to suppose. One saw cases—and I saw one or two which had been under Mr. Gunn's own observation—in which a bad prognosis had been given because of appearances denoting severe, or fairly advanced, arterial changes, possibly not pure sclerosis as was then thought, in which the opinion given was not justified by subsequent events; the patients lived for several years, and their health improved in many ways. There is no doubt whatever that Mr. Bardsley is right in his assertion that there are changes which, ophthalmoscopically, are not very different from those of arteriosclerosis but which have not nearly such a grave significance, and that if we observe carefully enough—as carefully as he has done—we shall be able to distinguish, at all events in very many instances, cases of true arteriosclerosis from those in which the appearances are due to abnormally high arterial tension.

Mr. D. LEIGHTON DAVIES (Cardiff): A short time ago I was engaged in examining the fundus in all cases of cerebral hæmorrhage at the workhouse at Cardiff, and I was struck by the very slight signs shown of retinal arteriosclerosis. It was very seldom that I found advanced signs. One of the commonest features I have come to look upon as indicative of arteriosclerosis is, not so much the blocking or indentation of the veins, as the redness of the disk: this redness was present in all those cases. And there was a certain indistinctness of outline in the arteries, not amounting to a difference of calibre. Here and there it was difficult to say where the margin of the artery ended, and where the retina started. But the point to which I should like to draw attention is, that in these cases we seldom see very marked indentation of veins. I took the blood-pressure frequently, and the systolic was often well over 200 mm. of mercury, and the diastolic 140 mm. Those persons obviously had generalized sclerosis: they were old people aged 60 to 70. It is very difficult to make any forecast as to the duration of life. Yesterday I saw the widow of a patient whom I examined in 1913 because of slight obscuration of sight. His age was 66, and he had worked very hard. He had slight signs of arteriosclerosis. In his case there was obliteration of the outline of the arteries, redness of disk, and slight tortuosity of small branches. He died in three years.

I told his medical man he might have a stroke, and he had three strokes last Sunday. Yet his blood-pressure was not very high, only 155 mm. There are cases which have been on one's book ten years with the same symptoms, and yet the patients still remain well. So it is difficult to discriminate between the cases that are likely to do badly and those that will probably keep well.

Mr. BARDSLEY (in reply): This paper was written nearly three years ago, and at that time I broke down in health. I have not read Mr. Foster Moore's contribution, but I shall have great pleasure in doing so.

Section of Ophthalmology.

President—Mr. William Lang, F.R.C.S.

(*June* 13, 1917.)

DISCUSSION ON THE ÆTIOLOGY AND TREATMENT OF IRITIS.

Opening Address by the President.

At the present time, when everything is in a state of flux and change, I think it will be of service to review the ætiology and the treatment of iritis. If we all agree as to the cause of disease in the majority of the cases of iritis we shall be in a strong position, should it appear politic, to advise the proper authorities to introduce measures which would prevent much ill-health, great loss of sight, and untold injury to the community.

To ascertain the causes which I found in the last 200 cases of iritis occurring in my private practice I have made a list of these causes and the number of cases assigned to each. They were :—

	Cases	Percentage
Syphilis	12	6·0
Gonorrhœa	24	12·0
Tubercle	22	11·0
General affections ...	17	8·5
Local affections ...	51	25·5
Pyorrhœa	74	37·0
	200	100

Syphilis, which was formerly thought to be the chief cause of iritis, occurred only in 6 per cent. of the cases—in hospital practice it would

probably be higher—but I think we may look forward to a time when it will be the rarest of the causes, thanks to modern methods of treatment, which make gummata of the iris, and of the ciliary body even, melt away without, as formerly, disorganizing the eye.

	Male	Female	Aged
Syphilis ...	12	— ...	22 to 60
Gonorrhœa ...	22	2 ...	22 to 53
Tubercle ...	10	12 ...	2 to 50; 20 cases between 16 and 35

Gonorrhœa, with its 12 per cent., is higher than is generally conceded, but in a general hospital it would be higher still. The description by Mackenzie of gonorrhœal iritis with its attendant rheumatism is still a classic, and until quite recently no advance had been made on his method of treatment, but since the infection has been found to persist in some portion of the genito-urinary tract for an indefinite number of years, relapses, the chief feature of the disease, can be prevented by local treatment applied to this source.

For this advance we must thank the surgeons who have paid attention to these incompletely cured cases of gonorrhœa. We now know that as long as the joints, tendons and fasciæ are painful, and attacks of iritis occur, there is some point in the genito-urinary system which is still affected.

It should be remembered that other sources of infection may complicate these cases, such as pyorrhœa, which can cause a relapse; also that organisms, other than the gonococcus, may be present in the genito-urinary tract.

Tubercular iritis was present in 11 per cent. of the cases: ten in males and twelve in females. It occurred at the average age of 25, all except two being between 16 and 35. These exceptions happened, one in a child aged 2, with a discharge from the ear which may have complicated the disease, the other in a woman aged 50, who, in addition to a well-marked tubercle of the iris, said she suffered from having an ancestor who had been cursed by a saint with special reference to the eyes, and ever since some member of the family had been blind.

The treatment for tubercular iritis is the same as for tubercle of the lung—good air, graduated exercise and food rich in fats, as well as local measures to subdue inflammation and prevent closure of the pupil.

GENERAL AND LOCAL AFFECTIONS.

Gout, 7; diabetes, 5; herpes of the fifth nerve, 3; influenza, 1; pneumonia, 1.

Skin, 10 (ulcers, 4; acne, 2; eczema, 2; boils, 1; insect bite, 1).

Mucous surfaces, 10 (middle ear, 1; frontal sinus, 1; antrum, 1; lachrymal sac, 1; tonsils, 6).

Peritonitis, 1.

Alimentary tract, 23 (gall-stones, 1; dysentery, 1; appendicitis, 1; colitis, 3; constipation, 17).

Genito-urinary tract, 7 (cystitis, 2; after each confinement, 2; vaginal discharge, 1; prolapsed and ulcerated uterus, 1; myoma, 1).

Injury, 1.

Sympathetic ophthalmia, 1.

Seventeen cases occurred with gout, diabetes, herpes of the fifth nerve, influenza and pneumonia. Ten were associated with some septic focus in the skin, or a mucous surface or cavity such as the middle ear, frontal sinus, antrum, lachrymal sac. The tonsils were affected in six. An affection of the alimentary tract was found in twenty-three, such as gall-stones, dysentery, appendicitis, colitis and constipation. The genito-urinary system provided seven—cystitis, perineal fistula, associated with each confinement, discharge from the vagina, ulcer of the uterus in a case of prolapse, myoma. A smart blow on the eye caused one case and a sympathetic ophthalmia another.

PYORRHŒA—24 MALE, 50 FEMALE.

Years	Cases			Cases
10 ...	1	...	Pyorrhœa and eczema	2
20 ...	5	...	,, ,, diabetes	2
30 ...	14	...	,, ,, gout	2
40 ...	15	...	,, ,, herpes of the fifth nerve	2
50 ...	26	...	,, ,, tonsils	3
60 ...	9	...	,, ,, constipation	8
70 ...	3	...	,, ,, appendix	1
80 ...	1	...	,, ,, ulcer of the uterus ...	1
			,, ,, tubercle	1
	74			22

Pyorrhœa was the sole cause to be found in seventy-four cases. When the patient was seen at the beginning of a first attack, the rapid recovery that took place after the removal of the affected stump, tooth or teeth, was very striking. No other treatment except atropine and heat was used. Twenty-two of the other cases had pyorrhœa as a complication—if these are added, 48 per cent. had their mouths affected.

In tabulating the cases where pyorrhœa appeared to be the sole cause of the iritis, it is interesting to note that there were twice as many women as men, and that the age incidence was very slight before 30, from 30 to 60 it increased, with a very sharp rise between 50 and 60, and almost ceased after 60. It will be of great value and interest to hear what our confrères of the Section of Odontology have to recommend as a preventative of this appalling state of affairs, which, starting in early childhood, pollutes the individual throughout life, or as long as he has any teeth in his jaws—all the time laying the seeds of numberless diseases in every part of the body, and many others in the eye beside iritis; in fact no structure of the eye escapes.

Sir Anderson Critchett, Bt., C.V.O.

With the very limited time at my disposal, perhaps my few remarks will be more useful if I confine myself practically to the question of the treatment of some of these cases of iritis.

I dare say many will remember, nearly forty years ago, that Sir John Tweedy made the suggestion that in many instances where atropine proved ineffective, the sulphate of duboisine was a more powerful mydriatic, and in some cases I have found that where atropine was practically powerless, I have been able to break down synechiæ with that still more powerful drug. It is never necessary to use more than 2 gr. to the ounce, and that is the practical equivalent of 6 gr., or even 8 gr., of atropine. It is important to be careful that it does not go down the duct into the throat, because in some instances the toxic effects may be rather alarming. Then, I am old-fashioned enough still to believe that leeches, in the early stages of acute cases, are extremely useful; and in the more chronic cases, keeping small open blisters on the temple is sometimes of very marked benefit.

I see that the President has alluded to the question of gonorrhœa as a cause of iritis. Undoubtedly it is a very frequent cause, and I would like, here, just to pay a little tribute to the memory of my old colleague and pupil, John Griffith, who was one of the first to follow out suggestions of Mackenzie, and who read an excellent paper on this subject. When he died, we lost a very valuable member of the ophthalmic world.

Then I come to the question of the advantage or disadvantage of iridectomy. Some thirty years have passed since Mr. Nettleship

introduced this subject. Those thirty years have gone and, I am sorry to say, our distinguished colleague has gone with them. The following is a short extract from the paper which he then read. With reference to iridectomy he said :—

"Making every deduction for sources of error, and allowing also for the proneness of patients to attribute benefit to any active treatment, I feel little doubt that iridectomy does confer real good in some cases of iritis, and this whether an operation be undertaken during the active inflammation, or in the intervals between the attacks. The benefit sometimes takes the form of a very long term of freedom, sometimes an increase in the length of the inter-iritis periods, and sometimes a diminution in the severity of the attacks."

He then goes on to say that the cases where, in his opinion, iridectomy is contra-indicated are those of keratitis punctata, the chronic thickening of the iris, with very extensive synechiæ and the existence of myopia. And these, in his opinion, were indications that iridectomy was not likely to be well borne, even if a good piece of iris could be removed. He finishes by saying :—

"I am, therefore, clear that it is our duty sometimes to advise iridectomy against recurrent and obstinate iritis. I feel sure that disappointment of both the patient and the surgeon would often be avoided if the operation be held in reserve until all other plans of treatment have been patiently tried."

With reference to my own rather long experience, I do not wish to speak in any exaggerated terms, but I think I may safely say this: that few things have impressed me more than the extraordinary value of iridectomy, both as a preventative and as a curative agent, in those cases for which it is specially intended. But, and where you have to deal with a half-rotten, friable, and in some instances "*bombé*" iris it is rather a big "but," the iridectomy, to be thoroughly effective, must be a good one.

As it is very necessary to have perfect quiescence on the part of the patient, I almost invariably give a general anæsthetic. I prefer to use a keratome, because I think it makes the cleanest cut, and because with it I think you can best measure the size of your wound. In introducing the iris forceps, I think it is necessary, in most instances, to go down almost to the pupillary margin, and then comes a gentle, almost coaxing, withdrawal of the forceps with a piece of included iris, and one clean cut with the scissors. I am old-fashioned enough to prefer the scissors that we used to have at Moorfields, set at right angles for the right eye, and curved for the left, though I believe many

men prefer De Wecker's scissors. I prefer to keep those for the operation for which they were originally intended—namely, iridotomy. If time permitted I could quote a very large number of cases in support of my belief in iridectomy, but I should like briefly to mention one case which has occurred in my practice within the last three years. A patient was sent to me by a general practitioner in the hope that I might possibly be able to save the last lingering portion of the remaining eye. The iris of the first eye was absolutely "*bombé*," and there was a closed pupil. In the second eye there was one small interval where I thought it was possible to remove a little piece of iris, and, to use a sporting metaphor, I "won by a short head on the post." Of course, I gave a general anæsthetic, and, I am glad to say, I got away a little piece of iris, leaving a quite respectable coloboma. The history of the case had been that repeated attacks had taken place over a period of four or five years, and the curious part, to me, was that the patient had been in the hands of a very able ophthalmic surgeon, whom I also knew to be an excellent operator. He is not here to-night, so no withers need be wrung. He had used atropine and perhaps other mydriatics: but he had watched this case go slowly from bad to worse. I have seen the case three times since, and I have heard from the general practitioner on several occasions, and, I am glad to say, no further attack of iritis has taken place in that eye. My first impulse was to write to the ophthalmic surgeon, but on second thoughts—and they are very often the best—I decided that he might think me guilty of swagger and impertinence, so I refrained. But it is just possible that the words I am speaking to-night may reach him through our *Proceedings*. Of course, he may still think he was in the right and I am in the wrong: but I am not without a lingering hope that if he should read these words and another similar case should come into his keeping, he may, perhaps, give a chance to iridectomy.

Mr. J. G. Turner.

The relation between diseases of the teeth and diseases of the eyes has interested me since the time when I worked at the Royal Westminster Ophthalmic Hospital as clinical assistant to the late John Griffith. Our object was to find out what influence septic teeth had on eye diseases. We had concluded that corneal ulcers were often very favourably influenced by extraction of such teeth when, to my great grief, John Griffith died.

It seems we are all agreed as to the infective origin of the vast majority of cases of iritis. The problem is to find the source of infection. The mouth, I believe, is far the commonest offender, though some rhinologists claim the horrid pre-eminence for the nose. In civilized communities there is hardly an individual who does not present some degree of dental sepsis. The less common sources of dental infection are abscess of the crypt of erupting wisdom teeth or of buried teeth in any part of the jaws, suppurating antra and cysts of dental origin, visible ulcers of the mucous membrane and gums. Far the more common are the marginal gingivitis and hidden ulcers of "pyorrhœa," the abscesses and chronic osteitis due to dead and septic teeth and roots, the inflammation and ulceration of the gums accompanying tartar, and the artefact inflammations and ulcerations due to rough and overlapping fillings and to badly made crowns, gold caps, and "bridges."

That dental sepsis is not more frequently recognized and incriminated is due, I believe, to its great prevalence. The abnormal is so common that it is accepted as normal. Its very prevalence makes many doubt its power for ill. More careful clinical instruction in the details of dental sepsis is urgently needed in our medical schools.

The tooth is the determining factor of dental sepsis. Infection may enter by way of the pulp cavity, following the track of dental caries; or by way of the tooth-socket, gaining entrance at the neck of the tooth, where the gum edge laps round it. When infection enters by way of the pulp cavity, once the root-canals are infected it is practically impossible ever to sterilize the root. Infection spreads radially in the minute dentinal tubules, and no means has ever yet been found of following up this infection and ensuring asepsis of the infected dentine. Infection spreads along the root-canals, and these are by no means always single straight canals running direct to the apex of a straight root. Often the canals are flat or dumb-bell shaped, or break up into branches or even a net-work towards the apex of the root, or turn off sharply at an angle to end near the apex, but not at it, as required by our cleansing instruments. The roots themselves are very generally curved, and the root-canals follow these curves. I have examined large numbers of "dead" teeth and roots after extraction, and I doubt whether I have ever found one tooth or root which did not show signs of having set up inflammation around it. There is either granulation tissue adherent to the root, exostosis of the root, or absorption of the root—all denoting that the root was abhorrent to the surrounding tissues. The plain fact is that when we are filling or

crowning a tooth that has once been septic we are taking our chances. An "eye for an eye and a tooth for a tooth" may be right, but an eye for a tooth is a foolish bargain!

When infection enters by way of the neck of the tooth, the first stage is the stagnation of germs, favoured by sticky food, on the neck of the tooth. Marginal gingivitis follows, and the gum edge which in its perfect condition should almost adhere to the tooth-neck, swells away from the tooth and opens a path for infection of the tooth socket. The now infected periodontal sulcus is an undrainable area, and tissue destruction proceeds at varying rates till pockets are formed, the alveolar bone being destroyed, leaving the gum still surrounding the tooth. (Where gum and bone destruction proceed at an equal rate the chances of infection are far less.) On the toothward side of this gum-flap there is always ulceration, however healthy the part may be to outward view. I have cut away large numbers of these gum-flaps and have always found ulceration on the toothward side, and it is, I believe, chiefly through these ulcers that infection, whether toxic or living, gains entrance. For a long time I tried unsuccessfully to stain bacteria *in situ* in these granulation-covered ulcers. At last I asked Dr. Plimmer to help me, and he at once stained organisms in the tissues of the ulcerated gum-flaps, showing their power for evil. Add to this that the pockets are filled with decomposing food and débris and we have here a further, and I think, an important source of injury.

If we accept the modern pathology of infection it seems clear to me that dental sepsis must be eradicated not only in cases of iritis but in the case of the large majority of diseases. I may here remind you that it is quite possible to live comfortably without teeth of any sort, and that tooth-plates may be a serious source of infection, since it is extremely difficult to keep them free from a greasy sour-smelling coating of germs and decomposing food.

Your President asks as to prevention. This is a very difficult subject. Cleanliness is the great preventive, both of dental caries and of "pyorrhœa." Unfortunately cleanliness of the teeth is extremely difficult to attain. The shapes and positions of teeth render the stagnation areas very awkward to reach. The usual brushing of the labial and buccal surfaces of the teeth is almost valueless, and the damage is done before the patient's attention is drawn to the deficiency of his methods. The details of tooth cleaning are too lengthy to enter into here, but some day it may be part of a nurse's (children's nurse's) training to learn how to clean teeth. If we could do away with sticky

flour we should do away with far the greater part of dental sepsis. There is also the possibility of extracting teeth to free the remainder, and so render them more "drainable." Unfortunately the best tooth for this purpose is the first permanent molar, the largest tooth of our set. The best age for extraction is between 16 and 20. Clinically, I have seen many cases where this tooth has been extracted at this age on one side allowing the remaining teeth of that side to space themselves. The extracted side at the age of 40 has remained free from caries and "pyorrhœa," while the unextracted side has been badly affected by both diseases. Unfortunately the explanation of this result is too lengthy to be entered upon.

Dr. Lyon Smith.

I feel great diffidence in taking part in a discussion on iritis with experts, but I have been specially interested in the subject because of its incidence in arthritic conditions, a subject upon which I have been making bacteriological investigations for the past eight years. The evidences which I have been able to obtain that bacteria or their toxins were the chief causes of arthritic conditions have been brought out in the study of vitiated conditions of the blood. I think most of these are brought about by bacteria, although I suppose, as Mr. Turner has just said, foods and other causes may alter the state of the blood. But at any rate so far as the prevention of this condition of iritis is concerned, there is one important element: bacteria and toxins prepare the ground, but it is the element of strain or injury which determines the site, whether it be a joint, a muscle, a nerve, or the eyeballs. So I have considered it sound practice to recommend every arthritic person to have his refraction carefully tested by an expert, and I think it is quite possible that a number of cases of iritis might be prevented if eye-strain were avoided.

For a long time people have supposed that tubercle bacilli, gonococci and the syphilitic virus were the chief organisms capable of setting up iritis or other inflammations of the eyeball; but, as we have just heard, the bacteria of pyorrhœa and those concerned with nasal catarrh, bronchitis, colitis, urethritis, salpingitis, &c., are all equally capable, either through direct infection by bacteria or by the circulation of their toxins or their ferments in the lymph, of giving rise either to conjunctivitis or to more deeply-seated inflammations of the eyes. In August, 1915, I published a short paper, giving an account of a test,

by direct hæmolysis, as a method of detecting bacterial toxins in the blood. The statements I made there have not been publicly controverted, so far as I know; I suppose that at the present time everyone is too much pre-occupied to test the truth or fallacy of the deductions which I then made. I have made over 450 examinations of various bloods, most of them from people who have been suffering from chronic rheumatism or gout. Amongst that number, I find there were eight cases in which iritis was a secondary trouble. A summary of the bacterial findings in these eight cases is as follows: The influenza bacillus or toxins were found in four cases, the toxins of tubercle bacillus in only three, and gonococci in two only. Toxins of streptococci and staphylococci were detected *in all* the eight cases, and *Bacillus pyocyaneus* was present in three of them. These two organisms—*Bacillus pyocyaneus* and the influenza bacillus—give off toxins which are nearly as depressing to the vitality of the body as those of tubercle. *Bacillus pyocyaneus*, for example, is a very common inhabitant of the alimentary canal in otherwise normal people. It is not easily detected in the excreta, nor in cultures from them, because it is usually smothered by coli organisms, which grow more readily. But in the blood tests of which I am speaking, it shows up very quickly, and a positive reaction is sufficient to give one a clue for a more elaborate search. The same holds true of the influenza bacillus, which produces a great variety of toxins that are specially prone to attack nerve tissues. I think iritis is a not infrequent sequel to a severe attack of influenza.

The only other point on which I have to remark is in reference to the treatment of these conditions by the addition of inoculations to the general treatment. If these are adopted—and I have seen them effective in more cases than I have quoted—it should be remembered that although in other parts of the body it is a good thing to set up a sharp reaction by giving fairly strong doses, yet when one comes to deal with eye conditions, marked reactions are too dangerous, because of the risk of precipitating a glaucoma. For example, with tuberculin, we should begin with a dosage of something less than the $\frac{1}{250000}$ mg. I have heard such a quantity described as negligible; it is scornfully stated to have no effect, one way or another. But if we investigate some experiments in colloid chemistry, we see how easy it is to demonstrate the effect of even billionth dilutions in entirely altering the character of a colloidal emulsion.

There was one other coccus I found, which was described as active in production of arthritis by Warren Crowe, named *Micrococcus*

deformans. It is certainly a very potent organism, because in a rheumatic person infected by it if you give even half-a-million of this coccus most violent reactions may follow. In two of the cases this organism was present, and a very small proportion of it was included in the compound vaccine which I prepared for the patients.

Captain A. W. Ormond.

There is one matter to which I would like to draw the attention of the Section. A challenge was thrown down some time ago as to rheumatic iritis, and I do not think it has ever been taken up. I noticed the President omitted it to-night. Does acute rheumatism—i.e., rheumatic fever—ever produce iritis at all? The argument is, that considering the thousands of cases of acute rheumatism which occur every year in London, and everywhere else, the number of recorded cases of iritis associated with acute rheumatism is negligible: I do not think there are more than two or three. Therefore in the cases referred to by the last speaker as "rheumatic," I presume he means they are so in the sense of having some arthritic condition, but not in the sense that they are due to the *Micrococcus rheumaticus*.

It appears to me that iritis is always the result of sepsis: due either to the organisms themselves or their toxins. If that be granted, it helps us very much in the matter of ætiology. I do not think it possible that any error of refraction or eye-strain can produce iritis.

The President mentioned, I am sorry to say, the question of gout. I do not know what gout is: I cannot make out, from books, that anybody has a clear idea what they mean when they speak of gout. Is it a bacterial condition? If so, I suppose it is possible that it can cause iritis. It does not seem clear whether gout is a microbial or a chemical condition. I am very sceptical indeed about gout ever causing iritis.

Another point about which I should have liked to hear opinions is, as to the length of time that the gonococcus can be demonstrated in the tissues. If, as has been claimed, the gonococcus remains in the crypts of the prostate, and similar places, for ten or fifteen years, and is capable of producing trouble after that interval, does that coincide with what is generally known of the life-history of the gonococcus in other tissues? I was talking with a genito-urinary surgeon this week, and he said the gonococcus could not live on indefinitely in the tissues.

If that be true, then these late cases must be due to something else, either associated with the gonococcus, or independent of it.

I have to thank Sir Anderson Critchett for his suggestions with regard to leeches. He spoke of them as an old-fashioned remedy, but I find leeches extremely useful; the difficulty consists in obtaining them. What I have also found extremely useful is the hot-coil: dry heat passed through a pad by means of a weak electric current; the heat is maintained at a uniform level, and the pad can be kept on continuously for twenty-four hours, or several days. It greatly relieves the pain of iritis.

Mr. Ernest Clarke.

I should like to make one remark in support of what Dr. Lyon Smith said. Captain Ormond said he did not believe that errors of refraction could be a cause of iritis. You cannot say that a badly-fitting boot is a cause of gout, but the imperfect boot ensures in the metatarsal joint of the big toe the manifestation of the gout which is already in the system. It is in that way I believe eye-strain operates in the onset of iritis. There may be many factors tending to cause iritis; but eye-strain may be the determining cause of the site being the iris. For the last twenty-five years I have been very careful to estimate the refraction of my patients with iritis when the attack has subsided, and I could bring you notes of many cases of recurrent iritis which have benefited enormously by the use of proper glasses: in the majority of those cases recurrence has been absolutely stopped. Some of the patients were going to submit to an iridectomy to stop attacks, but I have persuaded them to try glasses first. I do not say this to discountenance iridectomy, because that may, after all, be necessary, but to emphasize the importance of remembering the factor of eye-strain.

Dr. C. E. Lakin.

From the pathological point of view there are two important questions which need consideration. First, why should patients suffering with systemic infections develop iritis? Secondly, is such an iritis microbic or toxic in nature?

With regard to the second of these, I cannot recollect any instance in which the mere presence of a toxin was responsible for a local out-

pouring of an inflammatory exudate. An inflamed iris becomes discoloured for two reasons: first there is the increased vascularity attendant upon any inflammatory process, and secondly there is the presence of a more or less abundant exudate. I cannot at the moment think of any condition in which the mere presence of a poison is sufficient to determine the local exudation of an inflammatory fluid. So I strongly support the view that in iritis we have an actual microbic infection, and, moreover, we have an embolic process at work. That is to say, organisms entering the circulation from some quarter are carried by the blood-stream and are entangled as minute emboli in the iris.

We are now brought back to the first question, why should the iris be one of the spots picked out by these organisms? I think two reasons have been put forward. One is, that at the junction of the iris and ciliary body there is a change from a highly vascularized tissue to one relatively poorly vascularized, with the result that there is a diminution in available oxygen, and in consequence a diminished resistance to invading organisms. I do not know whether ophthalmologists can cite cases in which iritis has commenced at the junction of the iris and ciliary body, if so they would favour this view.

But I think the second view which has been put forward is well worthy of consideration. It is that the iris is vascularized by what to all intents and purposes is a terminal artery. We have the branches of the anterior and posterior ciliary arteries converging towards the pupil and forming an anastomotic circle. From this circle still smaller vessels are given off, which eventually form a network around the pupil. With such an anatomical fact in view, iritis, I think, becomes comparable to that condition which we sometimes see in young children who have received an injury to the lower end of the femur. I refer, of course, to that alarming series of events which we speak of as acute osteomyelitis or acute necrosis of bone. The main artery of supply to the femur after entering the nutrient canal divides into two branches, which make their way to the opposite ends of the bone, where they break up into a brushwork of minute vessels. When, after an injury, minute emboli of organisms, derived from some distant focus of infection, become impacted within this brushwork of vessels, acute osteomyelitis results. I take it very much the same thing happens where iritis supervenes upon a systemic infection.

The President, in his opening address, did not mention anything about quiet iritis. I refer to those cases in which the disease is not

infrequently overlooked because the patient is not aware of any redness or acute pain in the eye, but he gradually becomes conscious of failing vision, and so sees an ophthalmologist. On dilatation of the pupil it is perhaps found that synechiæ are present. I have wondered whether there is some very low form of infection in these cases, and whether the President has had experience of such cases in which he could demonstrate some low form of infection, perhaps less acute than in cases of iritis of ordinary severity. I have seen two cases of gradual optic atrophy for which I made vaccines; I do not know whether this has any bearing on the subject. The patients were suffering from advanced pyorrhœa, and there was unilateral optic atrophy in each case. The ophthalmic surgeon thought the gradual optic atrophy was due to the condition of the mouth. The teeth were extracted, and I made a vaccine from cultures taken from the roots of the teeth. In both cases the surgeon was able to report that the process had subsided, and although the sight was not improved, the process was no longer advancing. I wondered whether something of the sort might apply to cases of quiet iritis, whether there might be a fairly chronic infection of low intensity as a cause in such cases.

After what we have heard to-night there can be little doubt that the majority of cases of iritis owe their origin to the presence of some focus of infection in the body. This has been impressed upon clinicians by continued observation, and further, when the focus of infection is removed the eye improves. And it may be added that further evidence is forthcoming from animal inoculations. One observer inoculated twenty-two rabbits with *Bacillus pyocyaneus* intravenously, and afterwards irritated the cornea. In five he produced definite iritis, from which he was able to recover the infecting organism. Similar experiments have been carried out with streptococci.

Mr. Leslie Paton.

We owe to our President the recognition of pyorrhæa as a very potent source of iritis. Possibly it is in his honour that speakers to-night have laid a disproportionate stress on it as the most important cause. At the risk of increasing that disproportion, I will speak of a case in which a fairly direct connexion could be proved between a tooth-abscess and an attack of iritis. It was a case of very deep-seated keratitis profunda at first, associated later with considerable iritis.

When it came under my observation, there was a condition of onyx, considerable iritis with adhesions, and some hypopyon. The only source of infection found was a septic tooth. I operated upon this case, and got Captain Douglas to be present with me when the operation was done. We carefully sterilized the surface of the eye, and incised the onyx, from which he made cultures. Again sterilizing the eye, we removed some pus from the anterior chamber, from which he also made cultures. In both these cases he obtained a mixed infection of *Staphylococcus aureus* and *Micrococcus tetragenus*. The last-mentioned organism is practically only found in foul abscesses about the teeth, nasopharynx or lungs, and I think its presence in the hypopyon pus is as direct evidence as you are likely to get in a human being of the connexion between pyorrhœa and iritis.

The PRESIDENT (in reply).

I agree with all Sir Anderson Critchett's remarks as to treatment. I have learned a great deal from Mr. Turner's instructive speech, and if we follow his advice in treating patients, and tell them how to take care of themselves, we shall avoid many attacks of iritis, as well as many other diseases which come under the consideration of other Sections of our Society. There is no single part of the eye which is not influenced by sepsis. One thing upon which Mr. Turner did not lay stress is the importance of X-ray examination of the mouth in all cases in which there is doubt as to the existence of tooth infection. Some of the causes of obscure origin of iritis exist in the apices of teeth where the gums and the necks of the teeth appear to be normal, but a skiagram shows that the roots are anything but healthy.

Dr. Lyon Smith's information is most valuable: it will be a great help if by blood tests one can discover the cause of some of these obscure cases of iritis.

I also agree with Mr. Ernest Clarke's statement that it is important to correct any error of refraction. But I think it is still more important to try to eliminate any sources of sepsis; there is a natural tendency for all these cases to recover and then to relapse, and such tendency to relapse is materially helped by eye-strain. If, however, the source of infection can be removed, eye-strain does not matter at all, it cannot produce a relapse.

With regard to Dr. Lakin's remarks about "quiet" iritis, I am sure

he is right in his surmise that there is some special micro-organism associated with that variety of iritis. We are still in the Dark Ages as to the causes of an enormous number of cases of iritis. I do not doubt, however, that when we can devote more time to research work, we shall be able to recognize, by the clinical appearance of the disease, the site of infection, and shall be able to say, in a case of quiet iritis, that one must look for a certain microbe in certain places, just as in gonorrhœal iritis or in a syphilitic case when one sees a gumma on the iris there is no need to ask questions.

It is interesting to hear Mr. Paton's report of the recovery of a micro-organism from the interior of an eye. If we could get a few more such cases, we should be helped a great deal in solving the mystery.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

J. Y. W. MacALISTER

UNDER THE DIRECTION OF

THE EDITORIAL COMMITTEE

VOLUME THE TENTH

SESSION 1916-17

SECTION OF OTOLOGY

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1917

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Section of Otology.

President—Captain H. J. Marriage, R.A.M.C.

(*November* 17, 1916.)

Case of Chronic Adhesive Otitis: Myringotomy and Partial Ossiculectomy.

By P. Watson-Williams, M.D.

Miss S. E., aged 26, came under observation in May, 1916, with deafness of several years' duration. Membrana dry, slightly opaque and thickened, only slight retraction, with dry adhesive catarrhal otitis. Rinne negative. She had been treated in 1914 by another aurist, who repeatedly inflated the middle ear during four months, and ordered a nasal douche for post-nasal catarrh.

When I saw her first she could hear whispered words uttered as loudly as possible (H.W.W.), right 28 in., left 20 in.; after Eustachian catheterization (H.W.W.) right 54 in., left 36 in.

The maxillary antra and sphenoidal sinuses were explored by my suction syringe method, and muco-pus was found in the left sphenoidal sinus, which was opened on June 14, and at the same time the tonsils were enucleated. Subsequently repeated catheterization improved the hearing to H.W.W., right 88 in., left 38 in.

On September 20, Gellé test proved positive; I was led to the conclusion that there was absence of stapedio-vestibular ankylosis, and crucial myringotomy was therefore performed under nitrous oxide gas anæsthesia, with a view to determine how far direct exposure of the oval window to sound waves would improve hearing. Immediately after recovering from the gas anæsthesia the patient's H.W.W. was 7½ ft. (left). Apparently the middle ear was free from discharge, though naturally the bleeding prevented any careful inspection. Subsequently the patient developed a purulent otitis media, although every

precaution had been observed with a view to rendering the auditory meatus aseptic. When this had subsided, the patient's H.W.W. was right 6 ft., left 24 ft. On October 17 the left membrana was more freely excised, and the lower half of the handle of the malleus removed, with the intention of making the perforation of the membrana permanent. The beneficial effect of the simple myringotomy has been maintained, and now after further catheterization of the right Eustachian tube the H.W.W. is right 6 ft., left 24 ft. In other words, the so-called good ear is relatively the deaf ear, whereas the previously deafer ear has very useful hearing. Time will show whether the improvement, which is remarkable, will be permanent. The patient is submitted in the present stage with a view to opinion as to the desirability of deferring any similar measure on the right ear. It is suggested that the chronic dry adhesive catarrh of the middle ear on both sides was a latent infective process, without polynuclears, and probably due to the sinus infection. When the drum was incised, it was thought that the traumatism converted the latent into an active inflammatory infection, and it soon subsided under appropriate treatment.

It is open to any one to say that the otitis purulenta was due to defective precautions having been taken in the way of prior disinfection, and in regard to instrumentation; but I took the usual precautions, and I do not think that was the explanation. I think it illustrates the point which has been emphasized by Mr. Fraser—namely, the possibility of a dry adhesive catarrh being a latent infection, which may pass on to a labyrinthine process resulting in otosclerosis. Another point I suggest is the desirability of investigating the condition of the nasal sinuses, especially the sphenoidal, in cases associated with infection in the middle ear. In all my mastoid cases I investigate the condition of the maxillary antrum and sphenoidal sinus, and I think some of our disappointing cases are due to infection of those sinuses.

DISCUSSION.

The PRESIDENT: I should like later on to hear from Dr. Watson-Williams whether this patient's hearing remains good, or whether the present improvement was only temporary. I make this request because I have found so often in the treatment of these cases of chronic dry catarrh by operation, such as the removal of the drum and skin grafting, &c., that though the hearing improved for a time, afterwards sclerosis occurred, and the deafness became as marked as before.

Sir WILLIAM MILLIGAN: Dr. Watson-Williams is to be congratulated—at the moment at any rate—on a very excellent result; but we know these cases are apt to relapse. Many years ago I tried treatment more or less on the lines he followed in this case, but gave it up in disgust, because, although improvement took place and lasted a few months, I do not think I could name a case in which it persisted for over a year. Dr. Watson-Williams may still see the re-formation of a cicatricial membrane. It is true that amputation of a portion of the handle of the malleus militates against this, but, even so, I doubt if there will be permanent improvement. With regard to his explanation as to how the middle ear became infected, I regard it as far-fetched. No one can be quite certain of rendering the external meatus aseptic, and any one may have sepsis after such an operation—though I am sure Dr. Watson-Williams took all ordinary precautions. I do not think his conclusion that the middle ear was the seat of a latent infection is legitimate, because, according to Dr. Watson-Williams's own statement, when he opened the middle ear it was quite dry. [Dr. WATSON-WILLIAMS: It appeared dry.] At any rate, there was no secretion, and no exudate visible. The patient had had sphenoidal sinusitis on that side, and post-nasal catarrh—perhaps the result of the former—so we have not far to look for the source of infection. There may have been infection of the Eustachian tube, and with manipulation the middle ear became infected *per tubam*. I used to think that part of the want of hearing in these "dry" ears was due to the lack of lubrication of the vesicular chain. The case is, however, a good example of at least the temporary value of myringotomy.

Mr. HERBERT TILLEY: Most of us remember the great boom, some twenty years ago, of this method of operating for chronic adhesive catarrh of the middle ear. Papers recording their experience were written by Dr. Burnett and Dr. Sexton, of New York, special instruments were devised by them, and many ossiculectomies were carried out as a result of their former experiences. I performed the operation on a number of hospital patients, and on a fewer number in private practice, having clearly explained to each of them that without treatment there was practically no cure, but that this operative method was stated to give great relief. I was allowed to try it on the bad ear, the feeling being that it could not make the hearing of that ear worse, and it might make it better. For a time, and in a few cases, there was gratifying improvement, but I did not have a single case in which it was permanent. For the last eighteen years I have abstained from operating for this type of deafness. I agree with Sir William Milligan as to the probable mode of infection of the ear in this case, but I do so from another point of view. I do not think anyone who has been interested in suppurative lesions of the nasal accessory sinuses can help being struck by the rarity of suppuration in the ears of these patients, even though the chances of infection are so manifold. If you take 100 cases of chronic suppurative otorrhœa, in only about five, if you search the sinuses, will you find suppurative lesions in them. I have published one striking case in which I unwittingly cured a patient's chronic

suppurative otorrhœa by operating on his right maxillary antrum. He said the discharge in his ear had stopped, though it had continued for years. That is one of the few cases in which I remember there appeared to be any connexion between the two lesions. I do not know why the ear should withstand infection in these suppurative nasal cases, unless it is due to auto-immunization.

Dr. W. HILL: It is extraordinary how many men have taken up this method and then dropped it. Toynbee gave it up, not only because he obtained no great results, but because somebody got meningitis in a case from infection. Some of the ears in these cases will suppurate after puncture, whatever precautions you take: one need not go to the nasal cavities for reasons for this. When I first worked at otology I looked up the subject, and I had a communication from Professor Politzer. When I saw him, and asked him about it, he said, "I have done it, but it is not a bit of good." Berthold, Schwartze and Halle described improvements, and again in America the subject was taken up, as has been stated. Eighteen years ago, Dench, of America, published a large number of cases, and I think that in his book it will be seen that he recommends it now. But every one else whose opinion one values considers that the game is not worth the candle. The patients upon whom I have operated in two or three cases of the kind during the last ten years have been doctors' wives; probably their husbands had read up the subject and they were prepared to take the risk. I was very much gratified on one occasion by getting a result similar to that obtained by Dr. Watson-Williams, for directly the patient came from under the influence of the gas she exclaimed that she could hear, and expressed her great delight. This lasted for two days, and the good result was due to the blood having acted as an artificial tympanum. Then I tried glycerine, which seemed to act better, but later there was a relapse. Then the husband asked me to remove part of the drum membrane, with the malleus and the incus, which I did, and we used glycerine and wool as an artificial membrane. But the whole thing went back after a few months, and I think the last stage was worse than the first. There were many adhesions and impaction of the foot-plate of the stapes. In two or three cases the preliminary myringotomy improved the hearing very considerably indeed, but, on the whole, I have had no encouragement to go on with it. One operator, Faulder White, has written on this operation, and, I think, practises it at the present day, but I do not think he has any great following. I shall be glad should we find we can revise our judgment and our conclusions on this special subject, but I fear we shall be doomed to disappointment.

Dr. DUNDAS GRANT: I am afraid we do not know the conditions which lead some perforations to remain open when we want them to close, and others to close when we want them to remain open. As a rule, when a perforation persists, it is because the vitality of the patient, or that of the tympanic structures is lowered, as happens in tuberculosis, scarlet fever, &c. It is difficult to say whether the fact of suppuration having occurred in Dr. Watson-

Williams's brilliant case favoured the persistence of the opening, but perhaps that furnishes an indication of means of keeping perforations open. With regard to the reason why the infection should have occurred, if we regard it from the physical standpoint, we see that the mechanical conditions are entirely changed in the tympanic cavity when one opens the tympanic membrane: the pressure is different from what it is when the membrane is whole. Thus, when we force air up through the tube in blowing the nose, we are certain to produce increased compression of air in the tympanic cavity, without solid particles necessarily finding their way up. But when an opening is made in the tympanic membrane, actual material, or at all events microbic contents from the naso-pharynx will be driven up. In the same way the tympanic cavity will often drain much better through the Eustachian tube when a perforation has been made in the membrane, just as when a pipette already full is to be emptied, one has only to remove one's finger from the orifice for the fluid to run out. In this way it is easy for infection to have occurred here, even though Dr. Watson-Williams's asepsis in the meatus may have been perfect. A writer in the *Monatsschrift für Ohrenheilkunde*, professed to keep perforations open by repeatedly injecting liquid vaseline up the Eustachian tube, until it ran out through the perforation. I tried it, and I think it kept a perforation open for some time, but I could not maintain the treatment long enough to judge of its permanency. I congratulate Dr. Watson-Williams on his good result, so far.

Dr. URBAN PRITCHARD: May I go further back in the history of this matter, and remind you that Sir Astley Cooper began this operation for deafness? He happened to perforate a membrane, and got this result. Then it healed up, and, of course, he had done no good. He had so many patients rushing to consult him after that case, that at last he refused to see any deaf patients at all. I am a pessimist as regards this operation. I have seen so many measures tried that I do not think we shall ever arrive at anything like permanent success. This excellent case of Dr. Watson-Williams' is a little spoilt because of the suppuration. If you get suppuration, and thereby permanence, you may get some lasting good. But there are all the dangers of suppuration. I think that in six months' time this case will be *in statu quo*.

Mr. SYDNEY SCOTT: I would even go a step further than Professor Urban Pritchard, and warn aspiring performers of this operation that the *ultimate* effect on hearing may be that the hearing becomes worse than it was previous to the operation, in spite of marked temporary improvement. Dr. Watson-Williams has wisely borne this possibility in mind by operating on a patient with *unilateral* deafness. Deplorable consequences were known to have followed this procedure when applied to both sides. I think we should like to know what the condition is, say, this time next year.

Dr. P. WATSON-WILLIAMS (in reply): I wish to thank members for the interest they have shown and the remarks they have made. I feel with them the need for great caution in accepting results until time has shown what the

ultimate effect may be. This patient improved before there was any time for suppuration to influence the result: the improvement was so immediate that it could not have been due to suppuration; still, subsequent further improvement might be due to the suppurative process. With regard to Mr. Tilley's point, that he has seen many cases without infection of the ear, I would say that there are a good many cases of otitis media, especially the suppurative form, in which, if investigated, we should find the sinuses infected. I have had cases of acute mastoiditis in which cure resulted from operating on the sinuses, the ear being left alone. When the chief trouble is in the ear itself, our attention is not often enough drawn to the sinuses. I agree with Mr. Scott's point, though in this patient it is hardly a moot question as to whether the patient will be worse for the operative procedure, than if one had left her alone. I chose the worse ear for operation first, and I told the patient and her parents that I could not guarantee anything at all, and that they must be prepared to take it for what it was worth. In spite of the gratification of the patient, I have refused to operate on the other ear until time has shown that the result in the ear operated upon is completely satisfactory. I shall be glad to bring the result before the Section after the lapse of further time. I have no note of any tinnitus before the operation, and I do not think there is any now.

(*November* 17, 1916.)

A Further Case of Otosclerosis associated with Otitis Media.

By J. S. Fraser, M.B., F.R.C.S.Ed.

At the meeting of this Section in May I showed a case of otosclerosis associated with chronic purulent otitis media and cholesteatoma. You may remember that the microscopic examination was rather incomplete because the region of the oval and round windows had been destroyed by a saw cut. I now wish to demonstrate another case in which I have a complete series of microscopic preparations from both ears.

The patient was a woman who died on January 4, 1916, at the age of 63. She was married in 1881 and her husband tells me that she was very deaf at this time. The patient attributed her deafness to an attack of scarlet fever in childhood. Both she and her brother had scarlet fever and otorrhœa at the same time. Her brother recovered

his hearing, but the patient gradually became deaf. Her husband informs me that she always had a purulent discharge from the left ear and complained of severe tinnitus like the roaring of the sea. The noises also had at times a "beating" character. She sometimes complained of dizziness. As far as her husband knows there was no deafness in the patient's family. I have been unable to obtain definite information with regard to the presence of paracusis, but her husband thinks that she did hear better in a noise. The patient visited the Royal Infirmary about fifteen years ago, and also attended the Eye and Ear Hospital, Cambridge Street, Edinburgh, and had the Eustachian catheter passed. So far I have not been able to obtain any report regarding the functional examination of the ear. Except for attacks of eczema the patient appears to have enjoyed good health. The husband tells me that the patient had one child before marriage and that after marriage there was one miscarriage. For three years before her death she was an inmate of the Poorhouse at Craiglockhart, to which she was admitted on March 26, 1913. She suffered from gangrene of the left foot for a short time before death: the temperature was raised for the last fourteen days. Post-mortem examination revealed cerebral softening.

Microscopical Examination of Right Ear.

(1) The right external meatus is normal, but the drumhead is slightly thickened in the posterior part—possibly the result of former otitis media.

(2) *Middle Ear.*—The tubal part of the tympanic cavity appears to be wide. The mucous membrane of the tympanic cavity appears normal, except in the region of the oval window where the submucosa is thickened, congested and œdematous. The round window is healthy and the malleus and incus are normal. The stapes is ankylosed by bone to the anterior margin of the oval window. In the roof of the tympanic cavity the marrow spaces are congested and, where they lie next to the Eustachian tube, they show leucoblastic reaction or fibrous tissue. The tympanic muscles appear to be healthy.

(3) *Labyrinth Capsule.*—Above the level of the oval window there is an area of osteoporosis or ostitis vasculosa. Engorged vessels are seen entering this area from the deep layer of the swollen tympanic mucosa. The ostitis vasculosa shows a tendency to extend forward into the cochlear capsule between the cartilage and lamellar bone.

(4) *Labyrinth Contents.*—(*a*) Cochlea: Corti's organ is fairly well formed in the middle and upper coils, but the hair cells are not recognizable. In the lower coil the auditory papilla is not so well formed, but this is not unusual. There appears to be some shrinkage

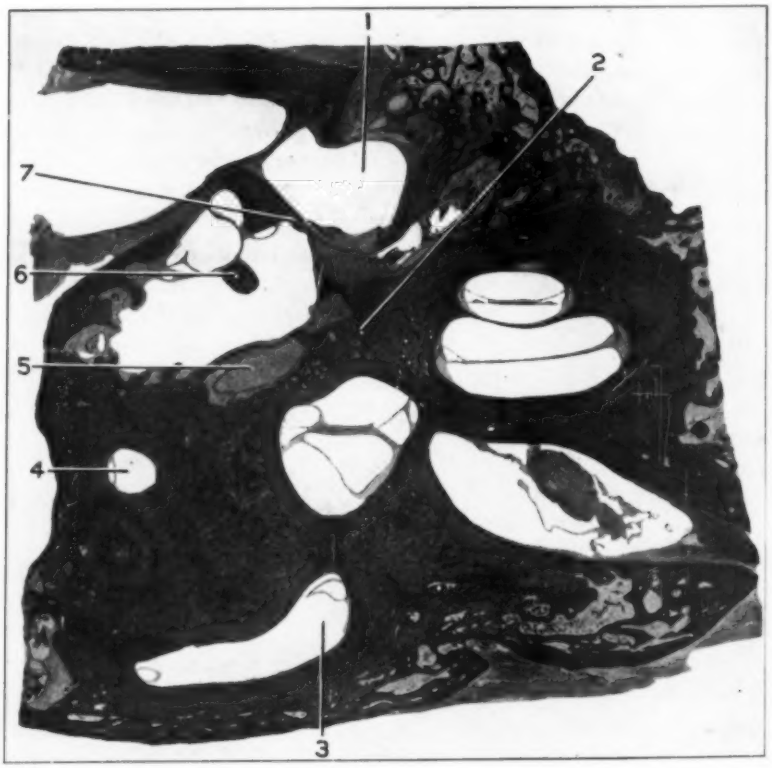

FIG. 1.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through right ear. No. 180. 1, anterior or tubal portion of tympanum; 2, area of ostitis vasculosa (otosclerosis) just above level of oval window; 3, crus commune; 4, lateral canal; 5, facial nerve; 6, incus; 7, tendon of tensor tympani attached to malleus. (× 6.)

of the cells of the spiral ganglion, especially in the basal coil, and slight small cell infiltration of the ganglion, but these changes are too slight and indefinite to have stress laid upon them. (*b*) Vestibule: The

otolith membrane of the utricle is separated from the neuro-epithelium, but otherwise the vestibular structures appear to be healthy. The lining membrane of the saccus endo-lymphaticus shows a peculiar appearance—the connective tissue beneath the epithelium lining being thrown into polypoid folds. (*c*) Canals: The cristæ of the canals are not so prominent as usual, but otherwise there is nothing to note.

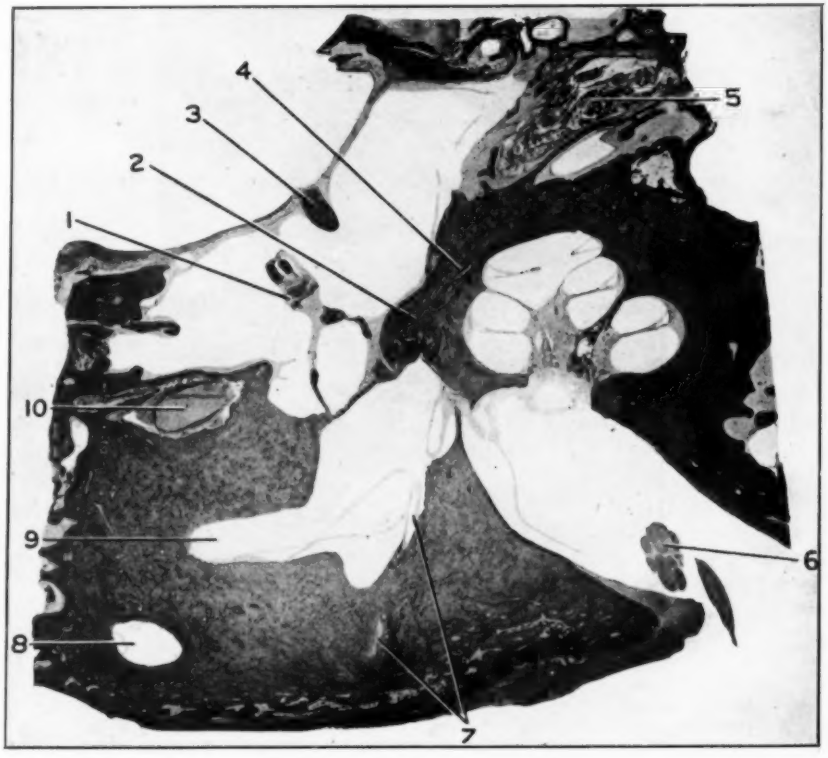

FIG. 2.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through right ear in region of oval window. No. 250. 1, joint between incus and stapes; 2, area of ostitis vasculosa in anterior margin of oval window; 3, Handle of malleus; 4 ostitis vasculosa spreads in cochlear capsule between cartilage and lamellar bones; 5, tensor tympani; 6, facial nerve: the eighth nerve is absent (artefact); 7, two parts of endolymphatic duct; 8, posterior canal; 9, smooth end of lateral canal joins vestibule; 10, facial nerve. (× 6.)

(5) *Internal Meatus.*—The eighth nerve has unfortunately been torn out in the removal of the specimen. The seventh nerve appears to be normal.

FIG. 3.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through right ear. No. 220. 1, thickened mucosa of anterior margin of oval window; 2, mucopurulent exudate in hollow of stapes; 3, posterior part of footplate; 4, region of ankylosis between stapes and oval window. Note the inflammatory process invading the anterior bony margin of the oval window from the deep layer of the swollen mucosa. (× 25.)

Summary.

There are slight evidences of an inflammatory change in the marrow spaces in the roof of the Eustachian tube and also in the mucosa lining the niche of the oval window. Elsewhere the middle ear structures are normal. The stapes is ankylosed to the anterior

margin of the oval window, and the bone in this region shows marked osteoporosis which extends forward for a slight distance into the capsule of the cochlea. Considering the age of the patient, the nerve structures of the inner ear may be considered to be practically normal.

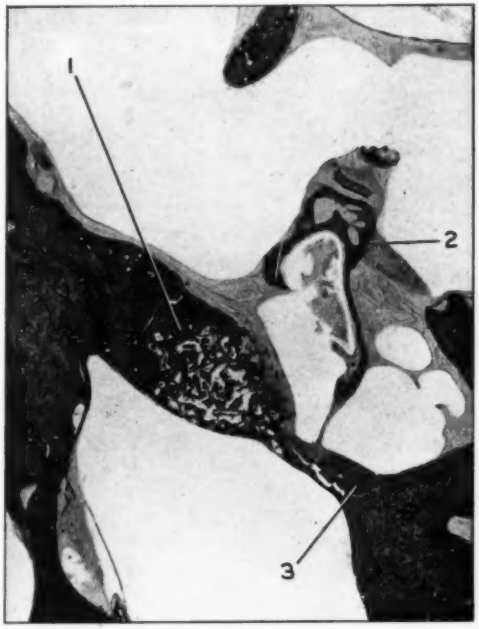

FIG. 4.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through right ear. No. 265. 1, area of ostitis vasculosa in anterior and lower margin of oval window; 2, tendon of stapedius attached to head of stapes; 3, split in bone of posterior margin of window (artefact). (× 12.)

Microscopic Examination of Left Ear.

(1) *External Meatus.*—The sub-epithelial tissue in the roof of the meatus is swollen and the epithelium itself desquamating. *Drumhead:* The tympanic membrane is very thick in its upper part and is bound to the inner wall of the drum cavity by adhesions. The lower part of the membrane is perforated: at the edges of the perforation the epithelium is absent. There is marked small cell infiltration of the

subepithelial tissue at the edges of the perforation. There is no extension of squamous epithelium from the outer surface of the drumhead round the edges of the perforation into the tympanic cavity.

(2) *Middle Ear Cleft.*—(*a*) Eustachian tube: The mucous membrane of the Eustachian tube is swollen and the submucosa infiltrated with small cells. A little pus is present in the tubal part of the tympanic cavity. The epithelium of the tube is clearly desquamated.

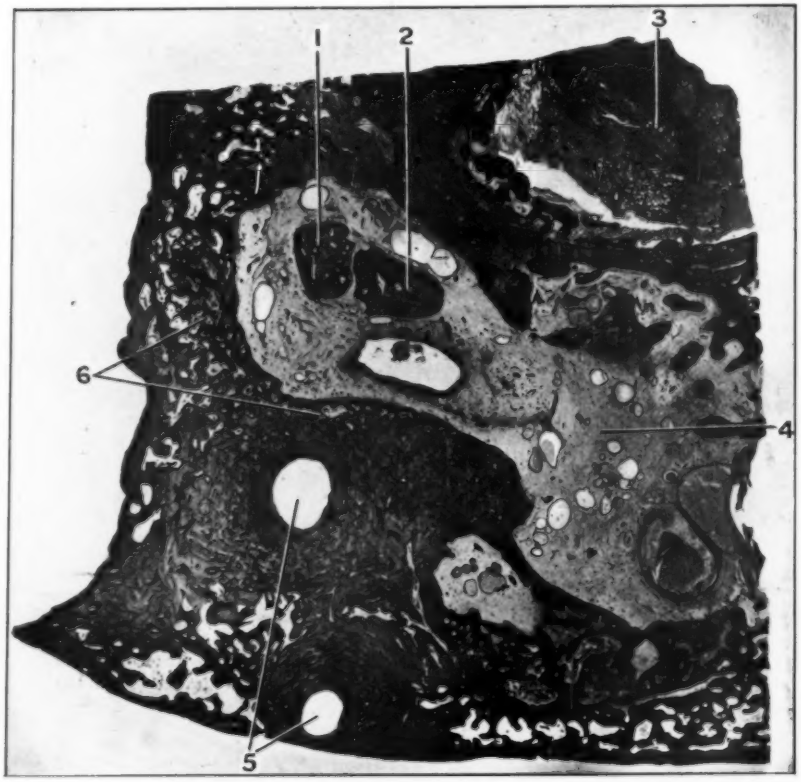

FIG. 5.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left ear in region of tympanic attic. No. 40. 1, malleus; 2, incus; 3, swollen lining membrane of external auditory meatus; 4, antrum filled up by swelling of submucous tissue; 5, two ends of the superior vertical canal; 6, areas of ostitis vasculosa in walls of attic due to inflammatory invasion of the bone from the submucous tissue. (× 6.)

(*b*) Tympanic cavity: There is great swelling of the mucosa of the attic, the submucosa being converted into granulation tissue. Cystic spaces containing pus are present in the attic and represent the remains of the attic cavity. The walls of these spaces show marked small cell infiltration. In the parts more remote from the attic cavity the bony walls show the usual fatty marrow, while nearer to the attic the marrow is more cellular and next to the submucosa the marrow spaces contain

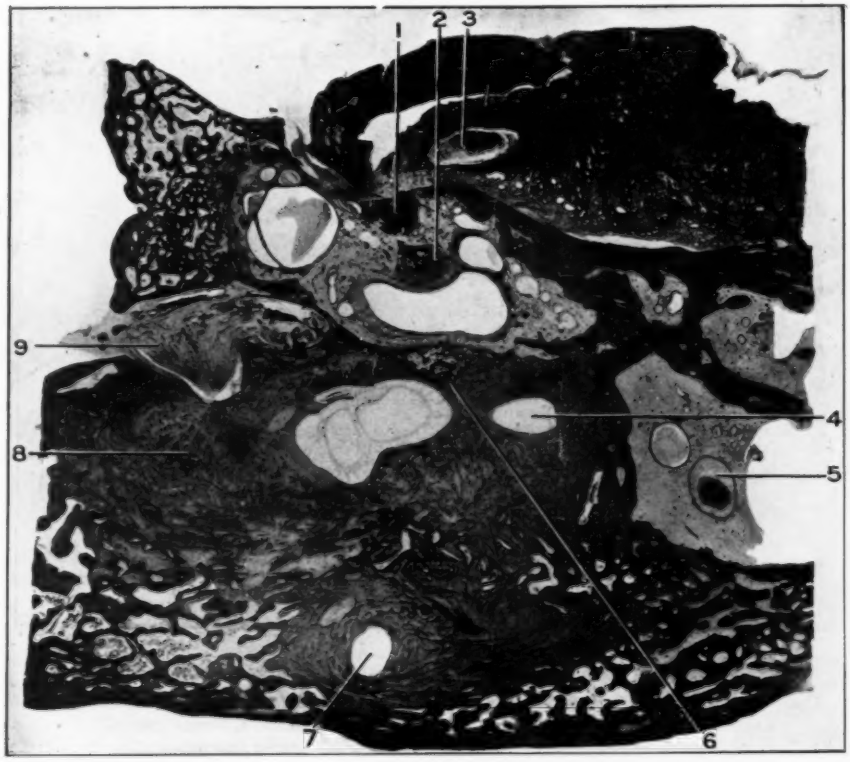

FIG. 6.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left ear above level of oval window. No. 105. 1, malleus (eroded); 2, incus (markedly eroded); 3, desquamated epithelium in external meatus; 4, lateral canal; 5, cystic space in antrum filled with mucoid material; 6, area of ostitis vasculosa just above oval window; 7, smooth end of superior canal; 8, capsule of cochlea; 9, geniculate ganglion of facial nerve. (× 6.)

granulation tissue. The bony walls of the attic cavity show marked osteoporosis and stain deeply with basic stains. The mucous membrane in the niche of the oval window is thickened, congested and infiltrated. The round window niche shows swelling of the submucosa with some

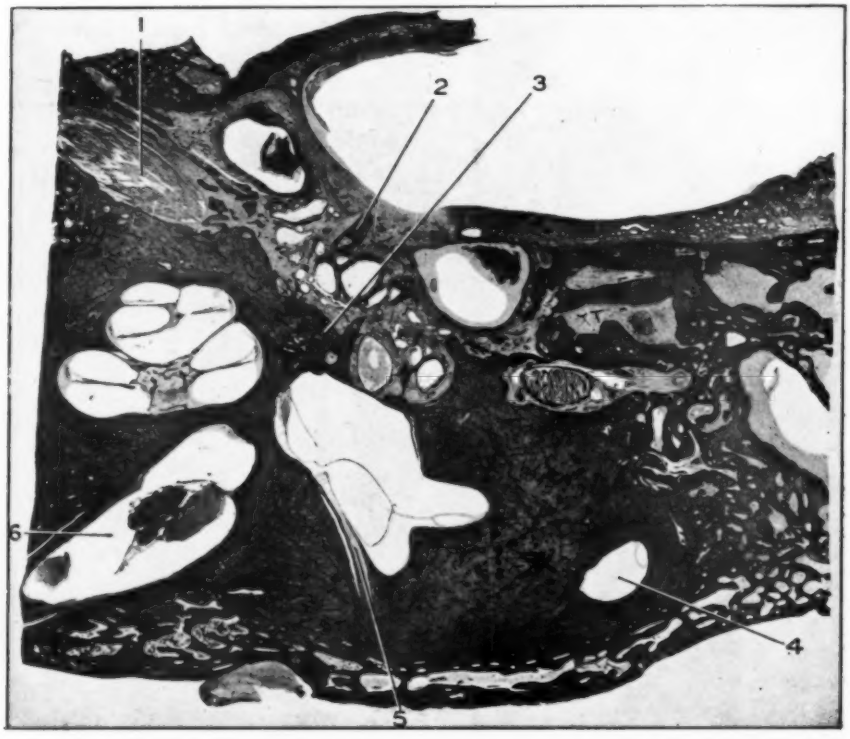

FIG. 7.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left ear in region of oval window. No. 210. 1, tensor tympani; 2, malleus handles; 3, area of ostitis vasculosa (otosclerosis) in anterior margin of oval window, with anterior part of stapes ankylosed to it; 4, posterior canal; 5, endolymphatic duct; 6, internal meatus. Note the marked adhesive process in the tympanic cavity. (× 6.)

cystic spaces and adhesions but no areas of osteoporosis. Ossicles: The malleus is almost normal. The long process of the incus is plainly eroded and in parts almost resolved into granulation tissue.

There is marked ankyloses of the stapes to the anterior margin of the oval window. The foot-plate itself is greatly thickened. The posterior part of the foot-plate is not ankylosed, but is jammed tight against the posterior margin of the oval window. Tympanic muscles: The tensor tympani appears to be rather more fatty than usual and the processus cochleariformis is eroded. The stapedius is healthy.

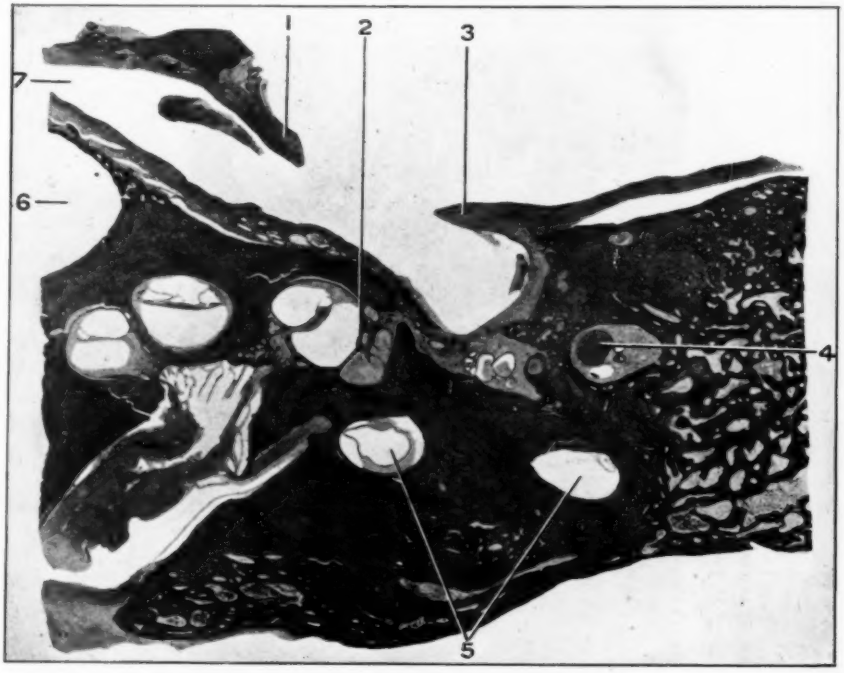

FIG. 8.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left ear in region of round window. No. 265. 1, anterior margin of perforation in drumhead; 2, membrane of round window; 3, posterior margin of perforation; 4, facial nerve; 5, two ends of posterior canal; 6, carotid canal; 7, Eustachian tube. Note that niche of round window and sinus tympani are both filled up by swollen mucosa. (× 6.)

(3) *Labyrinth Capsule.*—There is an *area of ostitis vasculosa in the bony prominence of the lateral (external) canal* as it lies on the inner wall of the auditus. The inflammatory change can be seen to pass in

from the congested and swollen muco-periosteum on the inner wall of the middle ear. There is a second area of ostitis vasculosa or osteoporosis in the anterior margin of the oval window, where the promontory is much affected. Below the level of the oval window ostitis vasculosa is still present and bulges inwards encroaching on the cavity of the vestibule. There is a third area of ostitis vasculosa at the apex of the cochlea just below the point where the tendon of the tensor

FIG. 9.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left ear in region of attic. No. 65. 1, external wall of attic; 2, malleus; 3, swollen submucosa; 4, ampullary end of superior canal; 5, area of ostitis vasculosa in bony wall of lateral canal; 6, remains of attic cavity; 7, short process of incus. (× 12.)

tympani is attached. (It has already been stated that the processus cochleariformis showed marked erosion of the bone.) Elsewhere the labyrinth capsule is healthy. The veins of the fossa subarcuata are dilated.

(4) *Labyrinth Contents.*—(*a*) Cochlea: Corti's organ appears to be well formed in all coils though the hair cells cannot be distinguished. The ganglion cells are very little, if at all, shrunken. The perilymphatic aqueduct is patent at both ends and is normal throughout. (*b*) Vestibule: The saccule and utricle with the endolymphatic duct are normal. (*c*) Canals: As on the right side the cristæ of all three canals are very low and the cupulæ are absent.

FIG. 10.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left ear just above oval window. No. 175. 1, malleus, 2, processus cochleariformis; 3, area of otosclerosis; 4, utricle; 5, facial nerve in shreds (artefact); 6, eroded long process of incus. (× 12.)

(5) *Internal Meatus.*—There are a few small cells in the fundus of the meatus. The vestibular ganglion appears to be healthy and the two divisions of the eighth nerve are normal. The usual corpora amylacea are present in the eighth nerve at the junction of the central and peripheral parts.

Summary.

On the left side the changes in the middle ear and labyrinth capsule are much more pronounced. Otitis media (purulenta) is still present and the drumhead is perforated. There is a marked chronic adhesive process in the upper part of the tympanum, with great thickening of the submucosa and erosion of the long process of the incus. The stapes is ankylosed to the anterior margin of the oval window. There are three well marked areas of ostitis vasculosa in the labyrinth capsule on

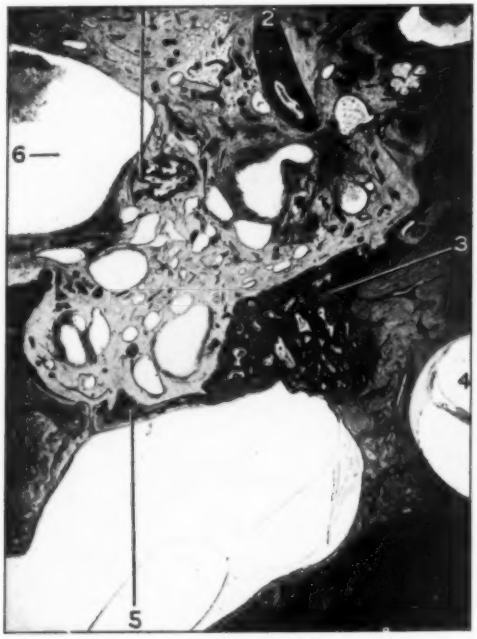

FIG. 11.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left oval window. No. 205. 1, long process of incus, showing ostitis vasculosa; 2, malleus; 3, area of ostitis vasculosa in promontory ankylosed to 5, footplate of stapes; 4, cochlea; 6, tympanic cavity. (× 12.)

the inner wall of the middle ear: (*a*) in the lateral canal; (*b*) in the anterior margin of the oval window; (*c*) at the apex of the cochlea. The labyrinth contents are normal.

I hold that in this case the otosclerosis on both sides was secondary to otitis media. On the right side the otitis media passed off but left traces in the region of the tube and oval windows. In time the inflammatory process invaded the bone in the latter area and resulted in otosclerosis with fixation of the stapes. On the left side the otitis media continued and invaded the labyrinth capsule in three distinct areas as stated above.

FIG. 12.

Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left cochlea. No. 245. 1, area of ostitis vasculosa at apex of cochlea in region of processus cochleariformis; 2, tensor tympani; 3, area of otosclerosis below oval window. (× 10.)

(*November* 17, 1916.)

Injuries of the Middle and Inner Ear in Fracture of the Cranial Base.

By J. S. Fraser, M.B., F.R.C.S.Ed.

At the present time it is hardly within the province of the otologist to enter into the general question of the method of production of fractures of the base. In the—let us hope near—future, when the otologist has developed into the cranial surgeon, it will be advisable for him to deal with the subject of "bursting" and "bending" fractures, and to indicate the direction of these fractures caused by falls on various parts of the head. At the present time it need only be said that fractures of the cranial base frequently involve the middle and inner ear. Brun[1] finds that there is disturbance of hearing in 14 per cent. of skull injuries and in 24 per cent. of basal fractures. The line of fracture may run parallel to the long axis of the petrous bone or at right angles to the long axis.

(1) Longitudinal fractures as a rule start in the orbit or region of the sella turcica, and pass backwards along the line of the middle ear cleft, breaking the roof of the Eustachian tube and tympanic cavity. The fracture may then pass outwards to the external meatus and squamous region: if this is the case the inner ear is not involved, though the ossicles may be dislocated and the drumhead torn. On the other hand the fracture after reaching the roof of the tympanic cavity, may pass inwards through the petrous pyramid, and thus resemble in some respects those fractures which run at right angles to the long axis of the petrous bone. The inner ear is of course involved in these latter cases. (Case II belongs to this second type.)

(2) Genuine transverse fractures of the petrous pyramid run at right angles to the long axis of the pyramid, and always injure the labyrinth. As a rule the fracture passes through the external meatus, roof of the tympanic cavity, vestibule and internal auditory meatus, as this is the line of least resistance. The fracture may, however, pass further forward through the cochlea, or further back when the canals are involved.

[1] Brun, H., "Der Schädelverletzte und seine Schicksale," *Beitr. z. Klin. Chir.*, 1903, xxxviii, pp. 192, 289, 601.

If the patient survives the injury at the time, he is not out of danger, as meningitis may supervene as the result of infection from the middle ear spaces, or of contamination of the blood in the external meatus and tympanic cavity. Such a complication is of course more likely if the labyrinth is involved in the fracture.

Stenger[1] has experimented on rats, in which he produced labyrinthine concussion by blows on the head. After the injury the rats seemed stunned and lifeless, but soon recovered. The animals were killed at various periods after the injury. In slight cases microscopical examination of the inner ear showed hæmorrhage confined to the region of the round window and lower coil of the cochlea. After more severe injury the whole of the cochlea showed blood extravasation and also hæmorrhages in the cochlear nerve.

From the point of view of microscopical anatomy, Nager[2] divides cases of labyrinth injury observed in the human subject, as the result of fracture of the base, into (1) recent and (2) old.

(1) The former cases are those in which the patient dies soon after the injury. In these Barnick[3] has shown that where the labyrinth capsule is injured, we have marked destruction of, and hæmorrhage into, the inner ear. Even in those cases in which the labyrinth capsule is not injured, we meet with hæmorrhage in the inner ear and eighth nerve. The auditory nerve may be ruptured (Lange[4] and Linck[5]) and the crura of the stapes torn from the footplate.

(2) With regard to the old cases, it appears that up to 1909 at any rate—the date of publication of Nager's paper—only two cases had been microscopically examined: (*a*) Manasse's[6] patient had suffered from fracture of the skull fifteen years before death, and yet on microscopic examination the line of fracture was still clearly visible—partly closed by fibrous tissue and partly by new bone. The break traversed the stapes and vestibule as in Case II recorded below. Manasse found new connective tissue and bone in the vestibule and in the basal coil of the cochlea. The scala media was dilated and the sensory cells and nerves were atrophied. (*b*) Nager[7] himself has examined the ear of an old deaf mute whose loss of hearing was due to an injury of the skull sixty years before death. In this case the line of fracture was not

[1] Stenger, *Archiv f. Ohrenheilk.*, 1909, lxxix, p. 43.
[2] Nager, *Med. Klin.*, 1909, v, p. 1525.
[3] Barnick, *Archiv f. Ohrenheilk.*, 1897, xliii, p. 23.
[4] Lange, *Zeitschr. f. Ohrenheilk.*, 1907, liii, p. 37.
[5] Linck, *Zeitschr. f. Ohrenheilk.*, 1909, lvii, p. 7.
[6] Manasse, *Virchow's Archiv*, 1907, clxxxix, p. 188.
[7] Nager, *Zeitschr. f. Ohrenheilk.*, 1907, liv, p. 217.

evident. The canals were obliterated by new bone, and the vestibule and basal coil of the cochlea filled up to a great extent by the same substance.

Case I.

The first case I wish to show is that of J. P., a male, aged 54, who was admitted to the wards of Mr. Charles (now Colonel) Cathcart, at 2 p.m. on December 6, 1910. I am greatly indebted to Colonel Cathcart

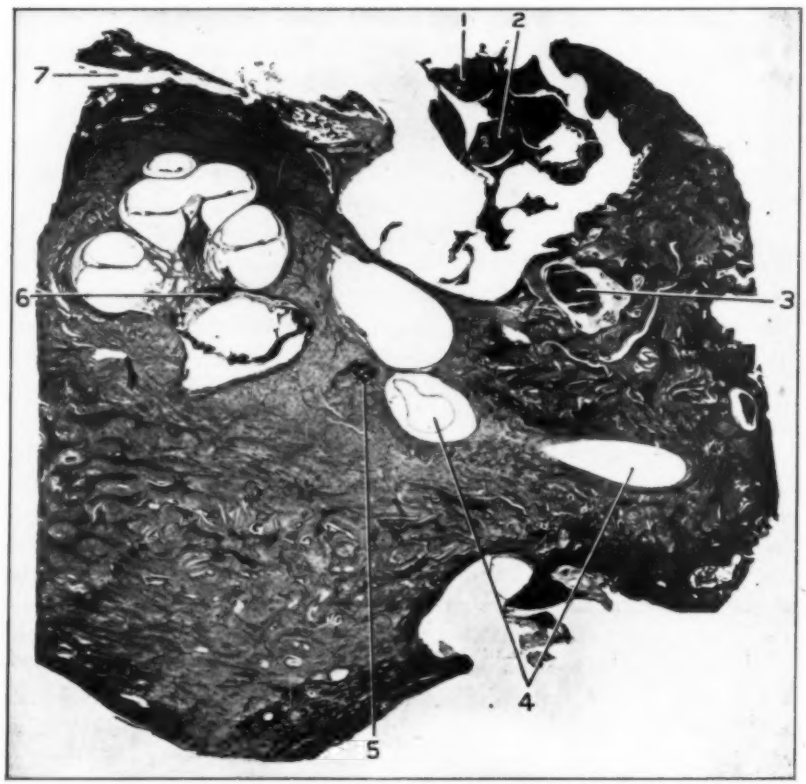

Fig. 13.

Fracture of cranial base without injury to labyrinth capsule J. P., male, aged 54. Horizontal section through right ear. No. 122. 1, malleus; 2, incus surrounded by blood-clot; 3, facial nerve; 4, the two ends of the posterior vertical canal; 5, hæmorrhage in branch of vestibular nerve to ampulla of posterior canal; 6, hæmorrhage in fundus of internal meatus; 7, hæmorrhage in Eustachian tube. (× 6.)

for his kind permission to record these cases. The history of the case was that, while stepping out of the way of an automobile, the patient was knocked down by a cable car. On admission to the infirmary the patient was pale, irritable and restless; pulse 64, temperature 96° F., respiration 28. Swollen bruised areas were present on the back of the scalp and above the left ear. There were no signs of injury to the body or limbs. There was profuse bleeding from the right ear, but *no flow*

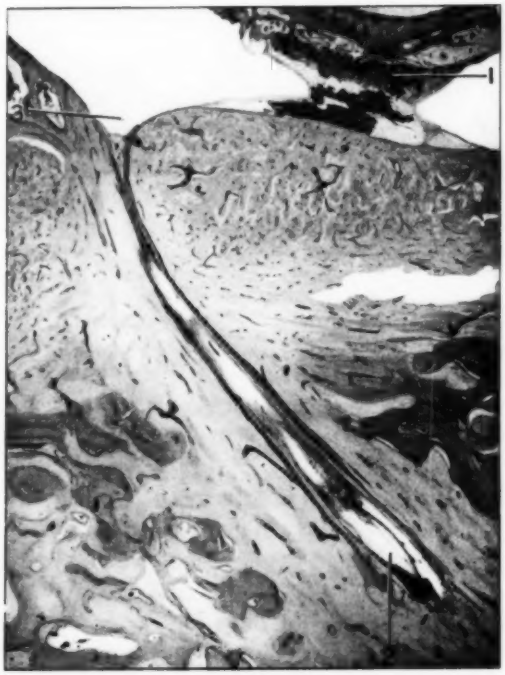

FIG. 14.

Fracture of cranial base without injury to labyrinth capsule. J. P., male, aged 54. Horizontal section through right ear. No. 170. 1, hæmorrhage in modiolus; 2, blood in perilymphatic aqueduct; 3, cochlear opening of aqueduct. (× 12.)

of cerebrospinal fluid (as will be seen later the labyrinth capsule in this case was not injured). The right pupil was more dilated than the left. Both reacted to light. Four hours after admission the patient became comatose and the breathing stertorous. The left arm was found to be

more flaccid than the right. Operation at 7 p.m. by Mr. Cathcart. The right temporo-sphenoidal region was trephined, but no extradural clot was found. On incising the dura dark blood escaped and a drainage tube was inserted. Breathing was quieter for a time after operation, but again became noisy at 11 p.m. At midnight, temperature 101·4° F., pulse 140. The patient died ten hours later.

Post-mortem.—The line of fracture runs along the roof of the middle ear cleft. The middle meningeal artery is lacerated and the roof of the tympanic cavity is splintered. The tympanum and mastoid cells are filled with blood. The jugular bulb is not injured. The fracture extends from the anterior part of the left middle fossa—where it crosses the groove for the middle meningeal artery—obliquely backwards through the squamous temporal and parietal bones to the lambda, and thence to the right parietal and squamous temporal regions. From here it passes down to a point behind the right petrous temporal, crosses the sigmoid sinus, and then runs along the anterior border of the right petrous bone.

Microscopic Examination of the Right Ear.

(1) *Middle Ear.*—The tympanic membrane is ruptured in its posterior part. Hæmorrhage is present in the mastoid antrum and cells and also in the tympanic cavity, but the joint between the malleus and incus appears normal. Blood is present in the sinus tympani and in the niche of the round window.

(2) The labyrinth capsule is not fractured, but hæmorrhage is present in the region of the geniculate ganglion and the fibres of the seventh nerve are separated by blood. There is also some bleeding in the fossa sub-arcuata.

(3) *Labyrinth Contents.*—(*a*) The cochlea only shows slight hæmorrhages in the modiolus. The scalæ of the cochlea are free from blood, but there is a little hæmorrhage in the aqueduct of the cochlea towards its cranial end. (*b*) The hollow spaces of the vestibule are free from hæmorrhage, but there is a little blood in the ductus endo-lymphaticus just before it opens into the saccus. The stapes is normal in the oval window. (*c*) The canals show no hæmorrhage either in the peri- or endo-lymphatic spaces.

(4) *Internal Meatus and Nerves.*—Hæmorrhage is present in the fundus of the meatus where the cochlear nerves enter the modiolus. There are also bleedings in the vestibular nerve and its branches to the superior and lateral canals, the sacculus and the ampulla of the posterior canal (*see* illustrations).

CASE II.

The second case is that of a stone-mason (P. B.), aged 41, who fell from a scaffold on November 1, 1910, at 8.30 a.m. The depth of the fall was only 9 ft. The patient was brought to the Royal Infirmary by the police who stated that when found the patient was conscious but was bleeding from the right ear and complaining of pain in his right shoulder. Examination on admission showed that the face

FIG 15.

Fracture of cranial base with injury of labyrinth capsule. P. B., male, aged 41. Horizontal section through right ear. No. 1. 1, blood in perilymph space of lateral canal; 2, 7, 6 and 4, show line of fracture; 3, blood in ampulla of superior canal; 5, vessels of fossa subarcuata. (× 12.)

was pale and the pulse feeble, 92 per minute. There was a flow of blood and cerebrospinal fluid from the right ear. (Microscopic examination showed fracture of the petrous bone involving the labyrinth

and extending from the inner wall of the middle ear to the internal auditory meatus.) The patient was conscious but very deaf. Soon after admission coma supervened and the breathing became stertorous. A little later general convulsions came on, the eyes being turned to the right—i.e., conjugate deviation to the side of the labyrinth lesion. Later on squinting was noticed, the right eye looking farther to the right than the left one. The right pupil was more dilated than the

FIG. 16.

Fracture of cranial base with injury to labyrinth capsule. P. B., male, aged 41. Horizontal section through right ear. No. 78. 1, scala media of middle coil, with slight hæmorrhage; 2, blood in scala vestibuli of basal coil; 3, rupture of Reissner's membrane; 4, blood in spinal ligament; 5, blood below basilar membrane. (× 30.)

left. The left side of the face and the left arm became paralysed, and the respiration was of the Cheyne-Stokes type. Pulse now 76. Operation by Mr. Cathcart 11 a.m. The right temporal region was

exposed and a fissured fracture found running from the anterior part of the wound downwards and backwards. A large extradural clot was exposed by the trephine opening, but the bleeding point could not be located even when the trephine opening was enlarged. The external carotid was therefore ligatured and the bleeding stopped. A drainage tube was inserted into the operation wound in the skull. Saline was injected *per rectum*. At 3 p.m. the respirations numbered 30 per

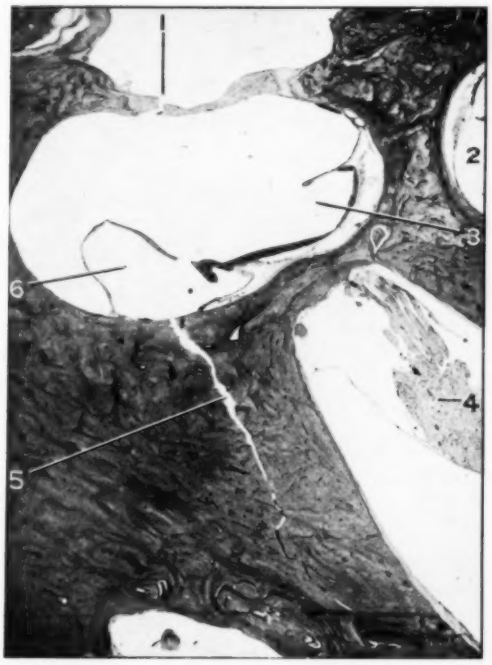

FIG. 17.

Fracture of cranial base with injury to labyrinth capsule. P. B., male, aged 41. Horizontal section through right ear. No. 125. 1, fracture through stapes; 2, basal coil of cochlea; 3, ruptured saccule with hæmorrhage; 4, vestibular ganglion; 5, line of fracture; 6, ruptured utricle. (× 12.)

minute and the pulse 128; temperature 100·6° F. The patient gradually got worse and died eight hours after operation.

Post-mortem. — Skull thicker than normal: brain œdematous; internal carotid arteries thickened. There is a considerable amount

of clot behind the trephine opening and the brain has not expanded. A fissured fracture is present passing from the parietal bone to the squamous temporal and then along the roof of the tympanum. No laceration of brain. The bleeding came from the middle meningeal artery (posterior branch). (Subsequent microscopic examination showed that the fracture must have bifurcated, one limb passing along the roof of the middle ear cleft and the other passing inwards

FIG. 18.

Fracture of cranial base with injury to labyrinth capsule. P. B., male, aged 41. Horizontal section through right ear. No. 175. 1, blood in scala vestibuli where it opens into vestibule; 2, line of fracture; 3, opening of smooth end of lateral canal (with hæmorrhage); 4, opening of smooth end of posterior canal into crus commune (with hæmorrhage); 5, blood in air cell; 6, blood in endolymphatic aqueduct; 7, inner end of fracture; 8, fundus of internal meatus with hæmorrhage; 9, basal coil of cochlea with blood in it. (× 8.)

through the labyrinth.) In preparing the specimen for microscopic examination the outer wall of the middle ear, along with the malleus, incus and tympanic membrane became separated as they were already

loose. Blood was present in the Eustachian tube, tympanic cavity, mastoid antrum, and cells. The line of the superior canal can be seen through the bone marked out by the hæmorrhage inside. The right clavicle is fractured and there is blood behind the manubrium sterni. Both lungs showed several gummata.

Microscopic Examination of the Right Ear.

(1) As stated above the outer wall of the middle ear is absent. There is blood in many of the air cells which surround the labyrinth.

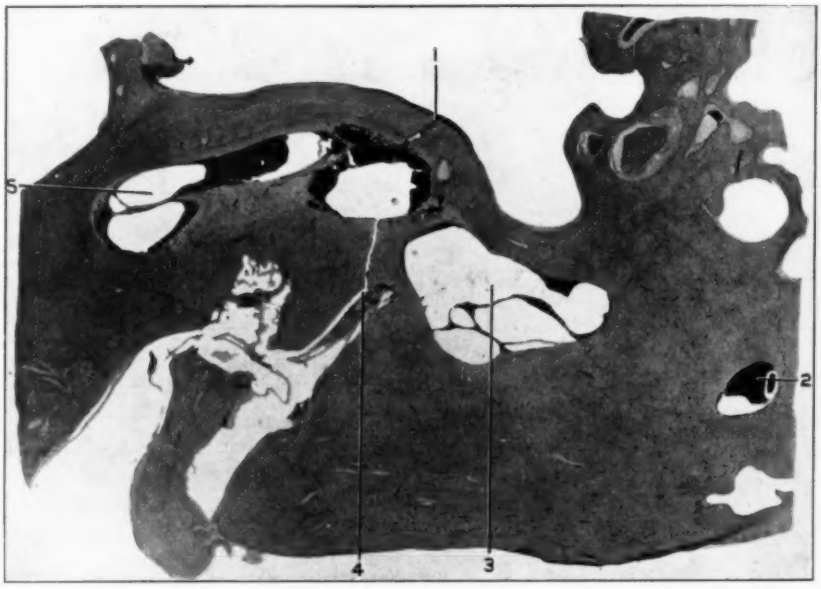

FIG. 19.

Fracture of cranial base with injury to labyrinth capsule. P. B., male, aged 41. Horizontal section through right ear just above round window. No. 235. 1, fracture of promontory; 2, both peri- and endo-lymph spaces of posterior canal contain blood; 3, vestibule with slight hæmorrhage; 4, line of fracture reaches canal for nerve to ampulla of posterior semicircular canal; 5, basal coil of cochlea with hæmorrhage in all three scalæ. (× 8.)

On the inner wall of the tympanic cavity there is some hæmorrhage present beneath the mucosa. Hæmorrhage is also present in the niche of the round window.

(2) *Labyrinth Capsule.*—The line of fracture passes through the superior and external canal, downwards through the oval window, involving the stapes and below this through the promontory. The fracture extends inwards and reaches the internal meatus but does not extend to the dura covering the posterior surface of the petrous bone. There is no hæmorrhage along the facial nerve in the aqueduct of Fallopius, although the bony walls of the aqueduct are involved in the fracture. The posterior crus of the stapes is broken. On passing

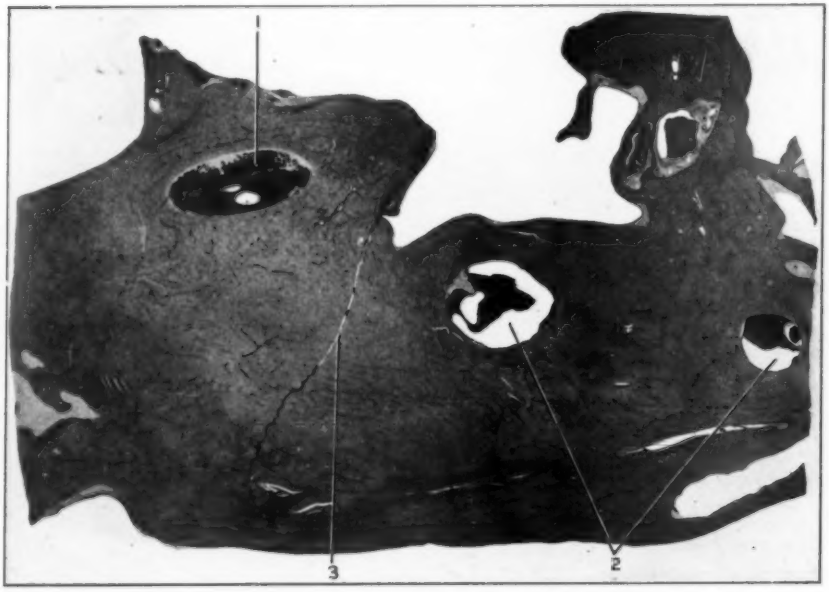

FIG. 20.

Fracture of cranial base with injury to labyrinth capsule. P. B., male, aged 41. Horizontal section through right ear below round window. No. 300. 1, extensive hæmorrhage in spiral ligament in lowest part of basal coil of cochlea; 2, both ends of posterior canal show blood in endo- and perilympth spaces; 3, line of fracture which does not quite reach dura mater of cerebellar fossa. (× 8.)

internally the line of fracture divides—one limb reaching the internal meatus and the canal for the nerve to the ampulla to the posterior canal, while the other limb stops short of the dura of the posterior fossa.

(3) *Labyrinth Contents.*—(*a*) Cochlea: There is marked hæmorrhage in the spiral ligament and in the scala tympani and vestibuli, especially in the basal coil. There is very little blood in the cochlear canal itself. In the middle coil there is no hæmorrhage except in the spiral ligament. There are a few small scattered hæmorrhages in the modiolus. The ductus perilymphaticus is free from blood. (*b*) Vestibule: The utricle and saccule are ruptured and there is blood in both the peri- and endo-lymphatic spaces of the vestibule. The ductus endo-lymphaticus also contains hæmorrhage. (*c*) Semicircular canal: Blood is present in the endo-lymphatic and peri-lymph spaces of the superior and external canals. The branch of the vestibular nerve to the cristæ of these canals shows extravasated blood. There is considerable hæmorrhage in the crus commune and in the peri- and endo-lymphatic spaces of the posterior canal.

(4) *Internal Meatus and Nerves.*—There is slight hæmorrhage along the vestibular nerve, especially along the branch to the saccule. The branch of the cochlear nerve to the basal coil also shows hæmorrhage. Although the fracture reaches the fundus of the internal meatus there is comparatively little hæmorrhage present in the meatus.

DISCUSSION.

The PRESIDENT: We are very much indebted to Mr. Fraser for these excellent specimens. Those showing the fractured base of the skull are particularly interesting to me. At a general hospital we are constantly seeing cases with fractured base, but usually not until some weeks after the accident, so that one cannot judge of the conditions immediately after the fracture; when they come to us, all that can be made out is, that they have complete nerve deafness. It would be interesting to know what is the effect sooner after the accident. For a long time I have tried to get the surgeons to let me know when the patients come in, but I hardly ever have the opportunity of seeing them in a recent stage. Having seen these specimens, I shall make a further effort to see if tests cannot be carried out immediately after the accident, instead of, as at present, weeks or months afterwards, as a collection of specimens with a complete examination of the hearing would be very valuable.

Mr. C. ERNEST WEST: I am obliged to Mr. Fraser for showing us these specimens. I should like to ask a few questions with regard to the case of otosclerosis. This woman was aged something over 60 when she met with her death. How long had she been deaf? How long does Mr. Fraser think the condition had been going on—if it had been going on at all? If it had not been going on, what was the exact nature of the resting condition? And what is it

that leads to progress in one case and to arrest in another? Histologically, are these patches of so-called ostitis vasculosa inflammatory tissue, or are they merely altered tissue—post-inflammatory tissue? I think that one very important point which Mr. Fraser brought out in connexion with his previous demonstration was the anatomical feature respecting the existence of a layer of what one might call cancellous bone really surrounding the true capsule of the labyrinth, and lying between it and what one might call the adventitious capsule, which is really a part of the petrosa. That seems to be the central part of his work so far as it affords a basis for the spread of an inflammatory and presumably infective process, whether it has penetrated from the tympanum or has originated by deposition, by blood infection. This case, in which there are apparently typical otosclerotic lesions on the one side and manifest results of past suppuration on the other, does lend colour to the theory that otosclerosis is essentially an infective or post-infective process originating in the tympanum. I reluctantly admit that I have been struck by Mr. Fraser's demonstration in that sense. I think it is important that we should arrive at a definition of what we mean by otosclerosis—whether a pathological process demonstrable histologically, or a clinical condition of a certain type as regards appearances, symptoms, and the like. At present we are a little liable to be at cross-purposes in our discussions, because we do not know whether we are talking of a pathological process demonstrable under the microscope, or of clinical types of deafness familiar to all of us. Before I sit down I should like to make a protest on the ground of accuracy in the use of terms. I am an upholder of exactness of phrasing as far as possible. The term I object to is "chronic adhesive process." I do not know what it means. I do not see any evidence for it in Mr. Fraser's slides. I see evidence of post-inflammatory conditions, but I see no reason why this should be called a chronic thing, any more than one's boot is chronic because it is always on one's foot. The condition is a resting one, and "chronic" to my mind implies a process which is going forward. It may be that there is a cicatrized tympanum—that there has been post-suppurative cicatrization. That is the way I should like to see it described. Ultimately, it seems to me, the turning-point of the discussions will hinge on the question of ætiology. When we have arrived at some clarified ideas with regard to ætiology, it is conceivable that we may arrive at some ideas as to treatment. I do not mean treatment when the patient has a developed otosclerosis, but treatment rather in the preventive or the abortive sense. It is extremely important that we should have laid before us, in as categorical a manner as possible, the views of those who are working on the subject as to whether this is a definitely infective condition—as to whether the process is due to some toxic or degenerative change taking place in tissue naturally prone to it, or whether we have to do with a chronic osteomyelitis. If so, there ought to be changes taking place progressively, and such appearances as Mr. Fraser has shown this evening in this woman aged 60 ought to be different from those in a patient aged 25 who is a progressive otosclerotic. That seems to me a point which Mr. Fraser should take up. There is one other point

that appeals to me, and that is the light which is thrown upon the formation of labyrinthine sequestra. One is familiar with the way in which the labyrinth is sometimes shelled out, with the inner layer of the capsule intact, over wide areas, not only in septic but in tuberculous cases, and we see a reason for that demonstrated in this cancellous sheath which runs round and over the ossified cartilage of the primary labyrinthine capsule. I have been leading nowhere, I fear, except to ask questions, but I am very grateful to Mr. Fraser for what he has shown us.

Dr. DAN MCKENZIE: I have always listened with pleasure to what Mr. West has to say, and it has been very interesting to hear from him that he has been convinced by to-day's demonstration that the inflammatory changes in the ear may lead to ostitis vasculosa. We have only seen two cases, however, in which this has occurred, and one would like to see more before arriving at definite conclusion, since both of these cases may simply have been coincidences. We do not know anything about their previous history; they are simply post-mortem specimens. This should make us wary about over-estimating their value in controversy. On the other hand, it is difficult not to believe that there is some connexion between inflammatory changes and these bone changes in the ear. It is not surprising that there should be such bone changes in inflammation of the mucosa of the ear. We must never forget that Politzer's typical otosclerosis, according to him and his fellow-workers, is characterized by the fact that in the mucosa of the middle ear there are no changes to be seen, and in the patient there is no history of former attacks of middle-ear trouble. It is difficult, I admit, to exclude all middle-ear trouble of infancy. I suppose very few of us have escaped such attacks, and yet the cases of otosclerosis among the generality of us would be insignificant. In other words, we have the association of a common occurrence and an uncommon sequel, and when this is the case there must be a third factor intervening to explain the sequel. It is true that another point in favour of the local cause is—what seems to be the case here—that one meets with cases of otosclerosis without any evidence of rheumatoid or arthritic trouble elsewhere. We get bone change precipitated locally in the ear in the temporal bone, and we naturally look for a local cause. The most obvious local cause is an inflammatory one.

Dr. A. A. GRAY (Glasgow): Mr. Fraser is to be congratulated on his results; it has been a great pleasure to inspect such beautiful specimens. Now, as regards Mr. Fraser's view as to the nature of otosclerosis, I am afraid I must say I am not entirely in agreement with him. I have examined half-a-dozen specimens of otosclerosis, and I have never seen any inflammatory change in the bone. I have never seen any round-cell infiltration nor any sign of inflammatory activity. It is difficult to accept the view of the disease being inflammatory when such a sharp line of demarcation is present. I mean that I can hardly conceive an inflammatory change stopping abruptly with the

sharpness of the otosclerotic focus. It rather gives one the impression of a tumour—a new growth. With regard to infection from the middle ear being the cause of otosclerosis, it is quite true that Mr. Fraser has shown us two cases apparently illlustrating this; but one must remember the number of cases which show the contrary. I have made pathological investigations of several cases of suppurative middle-ear disease, and I have not found otosclerosis in any of them; at the same time I know that the two conditions may occur coincidently, and I think that Mr. Fraser has happened to light upon two such cases. In these one must look for some antecedent tendency. Cases which fall into this category are not necessarily chronic suppurative cases; they may be cases of acute middle-ear inflammation. I recall such a case of middle-ear inflammation; it was very painful and acute, but healed without suppuration. The patient, however, to her physician's perplexity, continued to get more and more deaf. The explanation of the condition was that there was a marked tendency to otosclerosis in her family. The left ear—which was the side affected—gradually became more and more deaf, the right ear remaining perfectly healthy all the time. But, sixteen years later, she had an attack of appendicitis, and at the same time the right ear became slightly deaf and there was tinnitus; this was followed a little later by a second attack of appendicitis, and later by a third attack, after which the appendix was removed. Then the tinnitus disappeared, the degree of deafness remaining approximately the same. This was a case of acute middle-ear inflammation exciting otosclerotic change in the bone on one side; then, after the lapse of sixteen years, the other ear began to be affected. One might say that this was a case due to infection. It may have been infection from the appendix, but it was not from any acute or chronic middle-ear inflammation. In most, if not all, of the cases of otosclerosis I have clinically examined that have apparently started from suppurative middle-ear disease, on going carefully into the patients' histories and eliciting the facts and getting information as to deafness in relations, such as uncles and aunts, as well as parents and grandparents, I have almost always found evidence of some family deafness. I do not believe that otosclerosis can occur in the majority of individuals. It is true that we never know what we carry about with us in our body, but the majority of people could not be otosclerotics by any process whatever; and, on the other hand, there are a certain number of individuals who can never be prevented from becoming otosclerotics, because the tendency is so strong. And between these two classes of persons there are a number who will become otosclerotic under certain conditions which induce changes in the bodily constitution—e.g., typhoid fever, pneumonia, exhaustion, or various other conditions of that nature. In these cases otosclerosis is contingent upon this immediate condition, but the essential factor lies in the personal quality of the individual. The organ of hearing will withstand damage from various causes up to a certain point, but beyond this in some individuals it will give way, and in others it will not give way.

Mr. R. LAKE: At the International Congress of Otology, held at Boston in 1912, Professor Siebenmann (I think it was) showed a large number of photographs, and among them were some from patients who had suffered from suppurative otitis, in which these patches of osteitis were extremely large. I am inclined to think the case we are discussing this afternoon is due to a similar condition.

Sir WILLIAM MILLIGAN: I am not prepared to argue the point from microscopical appearances, because I have done so little work on otosclerosis along this line. But I think it will be difficult for Mr. Fraser to prove that otosclerosis is secondary to a septic condition of the middle ear. The number of cases of suppuration of the middle ear is legion, but the number in which otosclerosis is present as a complication is very small. We have, I think, to look to an entirely different ætiological factor as the causative agent; probably it will be found to be some anomaly in one or other of the internal secretions of the body. I have always been struck with the relation between anomalies of menstruation and otosclerosis. The time at which the otosclerosis first manifests itself in young girls is about that at which menstruation commences. I am not prepared to say it is a question of cause and effect; it is only a striking fact. There is a deficiency of lime salts in the blood of many of these otosclerotics; is that connected with the production of the disease? Against Mr. Fraser's view there are also the objective evidences from clinical examination. In many of the vast number of cases of otosclerosis there is no sign of there having been any middle-ear disease, nor any history of such either. I quite agree with Dr. Gray that we have closely to investigate the family history of these patients; if we do, we shall find some hereditary tendency, and most frequently on the female side. It may be a case of hereditary transmission of defect in internal secretion. There will have to be a great deal of work done by Mr. Fraser before it will be possible for him to convince the Section that otosclerosis is the outcome of a previously existing, or actually existing, suppurative otitis media.

Mr. FRASER (in reply): I am very much obliged to the members of the Section for the way in which they have received my communications. The President's remarks emphasize the need for the co-ordination of research. General surgeons tell us that in cases of fracture of the base the deafness only comes on after the patient has more or less recovered from his injury. I maintain that this is not so. The deafness is present all the time, but only becomes manifest when the patient recovers control of his mental faculties and finds that in one—more rarely in both—ears the hearing has been lost. I feel sure that if such cases were examined by otologists, these views would be found correct.

With regard to otosclerosis, the theory that I support—namely, that in many cases otosclerosis is due to an inward spread of otitis media into the labyrinth capsule—has met with a good deal of criticism.

In answer to Mr. West, I can only say that I am sorry that the clinical history of my case is so imperfectly known. Mr. West objects to the term, "chronic adhesive process." It is quite possible that a better term might be invented, but the words serve to indicate the conditions which exist in the middle ear when a catarrhal or purulent otitis media results in the thickening of the tympanic mucosa and the formation of adhesions. In some of these cases, at any rate, the old term "chronic catarrhal otitis media" is really more accurate as an inflammatory process is still going on. In other cases the term "cicatrized tympanum" would be more correct.

Sir William Milligan and Dr. Gray do not agree with me as to the inflammatory nature of the changes in otosclerosis. I have never maintained that otitis media is the "be all and end all" of otosclerosis. We have all suffered from head colds and a certain amount of tracheitis and bronchitis, and yet we do not all have chronic bronchitis or bronchiectasis. Most of us have had some indigestion and hyperchlorhydria at one time or another, and yet we do not all suffer from gastric or duodenal ulceration. Every case of catarrhal rhinitis does not settle down in the middle meatal region and result in nasal polypus formation. In the same way very many children have attacks of otitis media, and yet they do not all develop otosclerosis later in life. Undoubtedly there is another factor—the hereditary tendency to the disease. In susceptible people the region of the oval window is a *locus minoris resistentiæ*. If a child with a tendency to otosclerosis gets an attack of otitis media, I believe that otosclerosis is very likely to develop. Dr. Gray made some remarks last May which appear to me to show that he also was inclined to this view. There is also a growing amount of evidence to support the view that in certain families cases of otitis media are specially likely to develop intracranial complications. The only way to get rid of the hereditary tendency would be to forbid people to marry if there was a strong tendency to otosclerosis in the family. Koerner takes this line, but it seems rather a "large order."

If Sir William Milligan will read the conclusions to the article by Mr. Muir and myself in the November issue of the *Journal of Laryngology*, he will see that we do not claim that all cases of otosclerosis are secondary to otitis media. We believe that there is a "primary" variety which occurs with an apparently normal middle ear, and is usually seen in young women. In such cases the infection probably comes by way of the blood-stream, and the condition appears to be allied to osteomalacia. In the second class of case the otosclerosis is "secondary," and is due to an inward extension to the labyrinth capsule of an otitis media. In this variety the sexes are probably affected more or less equally. Lucae was of opinion that in chronic progressive middle-ear deafness it was impossible to draw a sharp line between cases of chronic adhesive process in the tympanum and those in which otosclerosis was present, and I believe that subsequent observations will still further support that view.

The most important question to decide is this: Is otosclerosis an inflammatory condition, or is it not? I believe that it is, but my opinion is not

of great value. It is a very different matter with regard to the opinion of Mr. Richard Muir, who is one of the best known pathologists in this country. Mr. Muir holds that otosclerosis is undoubtedly of inflammatory origin. Dr. Gray can see "no sign of inflammation" and yet in the osteoporotic areas we find rarefied bone, in the spaces of which there are dilated blood-vessels surrounded by numerous small cells. Giant cells are also found in many cases. These appearances bear strong evidence of an inflammatory character.

I have been asked why, if otosclerosis follows otitis media, it is always located in the anterior margin of the oval window. I have shown you that in the present case the area of otitis vasculosa in the left ear was much more extensive, and affected not only the anterior margin of the oval window, but also the bony prominence of the external canal and the apex of the cochlea. I think it was Politzer who showed that at the anterior margin of the oval window there is a vascular connexion between the vessels of the middle ear and those of the labyrinth capsule. Bruehl has also pointed out that this area is liable to special stress on account of the movements of the anterior part of the footplate of the stapes and the presence of the tendon of the tensor tympani muscle in this region.

Sir William Milligan believes that there is some connexion between the occurrence of otosclerosis and disorders of internal secretion. Well, of course, the ductless glands are all "the rage" at present. After failing with iodides, phosphorus, fibrolysin, otosclerol and radium, I am now doing my best to be in the fashion and am treating cases of otosclerosis with extracts of the ductless glands—pituitary, thyroid and suprarenal. As yet the duration of treatment has been far too short to enable me to give a report on the results. My own view, however, is that it is *most important to make sure that cases of catarrhal and suppurative otitis media in children really do clear up completely.* Such cases should be carefully watched and treated for a long time after the acute symptoms have passed off. It is to be hoped that the attention now being paid to the operative treatment of the nose, nasopharynx and fauces of young children may result in fewer cases of otitis media and a better recovery from this affection when it does occur. If this is so, we may expect to see a decrease in those cases of otosclerosis which are secondary to middle-ear inflammation. Vaccine therapy in cases of recurrent catarrhal otitis media may also be of use.

Since I have adopted the President's method of immediate skin-grafting at the end of the radical mastoid operation, and have observed the excellent results obtained, I have come to the conclusion that it would be quite justifiable to operate on cases of otosclerosis. I suggest that after the completion of the radical operation, the bony cap of the lateral canal should be removed without injuring the endosteum and a skin-graft applied at once. In this way a movable window would be supplied to replace the ankylosed oval window. The operation should, of course, in the first place be performed only on the more affected or deafer ear.

Dr. Dan McKenzie suggests that it may merely be coincidence that in

these two cases otosclerosis was associated with otitis media. If this is his opinion, I do not agree with him. In the present case the patient had only slight remains of otitis media on the right side, but on the left there was a marked adhesive process in the upper part of the tympanum, while a perforation was still present in the lower part of the drumhead. It was on this left side also that the osteoporotic changes in the labyrinth capsule were most marked. The bony ankylosis is also more extensive on the left side.[1]

There does seem to be a loss of trophic nerve influence in cases of otosclerosis as evidenced by diminished sensibility of the external meatus and drumhead, and diminution or absence of the secretion of wax. The question is, Which is the cause and which the effect? If otosclerosis is an inflammatory condition, it cannot be entirely due to loss of trophic influence, but it is quite possible that weakening of this trophic influence may be an important factor in the pathology of otosclerosis, indeed, it may be that this is the way in which the hereditary tendency to the disease manifests itself.

[1] [*Note.*—January 11, 1917: Since my return from London I have examined microscopically a *third* case of otosclerosis associated with chronic purulent otitis media. I hope to show this case to the Section at an early date.—J. S. F.]

(*November* 17, 1916.)

A Note on the Monochord, with some Illustrative Figures.

By W. M. Mollison, M.C.

Ear tests are of doubtful value, but some of those in common use are at least interesting, and serve to differentiate cases of deafness into two main classes. Of these tests that for determining the *upper tone limit* is perhaps the most interesting: it has always been doubtful what is actually the highest note appreciated by the normal ear, and I do not think it is important. What is of interest is that the upper limit is fairly constant and can be very accurately determined. The instrument which appears to me to give the most constant results is the monochord. It consists of a length of steel piano wire stretched between two points on a steel frame on which runs a bridge; this bridge grips the wire and so alters the effective length of wire which is to be thrown into vibration. Longitudinal vibrations are produced in the wire by rubbing it with resined leather (Tyndall) or with cottonwool soaked in spirit. The great advantage of the monochord is that the note produced depends on three factors only: (1) The length of wire thrown into vibration; (2) the calibre of the wire; (3) the material. These three factors are easily standardized, and so an instrument is produced which gives always the same notes and requires no complicated standardizing when finished as is the case with Galton's whistle.

The monochord has another advantage pointed out by Mr. Scott some few years ago; not only can the upper tone limit through air be tested, but also that through bone; this possibility led to the discovery that Rinne's test with high notes is always negative. The accuracy with which the upper tone limit can be defined is most striking; a difference of $\frac{1}{2}$ cm. in a length of 20 cm. is quite easily appreciated by the patient.

The results of the test are expressed in lengths of wire, the note from which the patient hears as a squeak; the result for hearing through air is placed above that through bone (the frame of the monochord being held on the mastoid process); thus for normal individuals the result is $\frac{13}{11}$ as an average.

The following are cases of shell shock or gun deafness and are chosen as typical of such cases.

	Age	Hearing for whisper or conversation speech		Monochord		Notes
1	21	4 to 5 ft.	3 to 4 ft.	16/14	17/14	In France fifteen months
2	37	1 to 3 ft.	1 foot.	17/14	16·5/14·5	In the artillery
3	40	2 to 4 ft.	3 to 5 ft.	16·5/15·5	16·5/15·5	Royal Field Artillery; always tinnitus after shooting
4	35	8 in. conversation	3 to 4 ft.	30/18	18/14	Shell shock and gun fire
5	26	A. c.	3 to 4 ft.	36/30	31/21	Shell shock
6	38	3 in.	4 in.	0/45	26/17	Buried by shell one month ago; previous middle-ear catarrh
7	24	8 to 9 ft.	2 ft.	0/0	0/0	Left eye destroyed by bullet; tinnitus before War; upper tone limit, ? 4,000
8	40	8 to 10 ft.	9 to 11 ft.	25·5/16	26/17	Eight months ago a high explosive shell destroyed both eyes
9	20	6 in.	3 in.	30/14·5	28/18	Lost both eyes from shell explosion
10	24	1 ft.	12 ft.	18/14	15/14	Tinnitus; knocked down by sandbags
11	44	1 in. conversation	6 in. conversation	0/18	32/18	Previous deafness; no shell shock; Rinne negative
12	46	4 ft.	2 in.	22/16	42/22	Tinnitus four years; much quinine in India
13	21	3 to 8 ft.	10 ft.	13·5/11	13·5/11	Had been exposed to very severe shelling; thirty a minute for half an hour
14	24	0	0	34/16	34/16	Functional dumbness and blindness cured by suggestion; with an electrical aid could hear quite well with left ear

(*November* 17, 1916.)

Traumatic Dislocation of the Incus, which was found lying in the Antrum in the Course of a Radical Mastoid Operation.

By W. M. Mollison, M.C.

C. P., aged 38, was exposed to a violent explosion in April, 1916. The explosion occurred about 12 yards from him on his left-hand side. He was unconscious for twenty-four hours; he then found he was deaf

in the left ear. Soon afterwards discharge began and has continued. He has some pain in the ear and slight tinnitus.

On examination in July, 1916, there was found a large perforation in the left membrana tympani posteriorly; there was foul pus coming from the middle ear. Hearing in the ear was *nil*.

On account of the foul otorrhœa, the radical mastoid operation was advised. This was done in October. The mastoid process was cellular, but contained no pus; the antrum was, perhaps, a little larger than normal but it contained no pus. Whilst curetting was being done in the antrum and towards the aditus to remove small spicules of bone due to the gouging, the incus was found lying quite loose and bare, and stained by powder (?). The radical operation was completed, the malleus was in position and the anterior half of the membrane affixed to it; a particle of powder (?) on a small granulation was lying over the stapes. The cavity was skin-grafted. The malleus and incus are shown.

(*November* 17, 1916.)

A Case of "Cerebellar Storm."

By W. M. Mollison, M.C.

Dr. Fawcett has kindly allowed me to bring forward these notes on a case which was under his care in Guy's Hospital.

Nurse P., aged 24, had a mild attack of German measles on March 7, 1916; she was apparently well in about a week, except for severe rhinitis. On March 16 she was walking in the hospital grounds when she felt sick and vomited. She had vertigo and vomited several times on March 16, 17, 18, and 19, and was quite unable to take any food at all. At the request of Dr. Fawcett I saw her on March 20. She lay in bed with her eyes closed, and through the lids the eyeballs could be seen moving. She felt very giddy, complained that objects moved from left to right and that she herself felt as if she were moving. There was marked vertical nystagmus with deviation of the eyes upwards and the quick movements of the eyes downwards.

Examination of the ears showed a little wax in the right, but a normal membrane with ? slight retraction; on the left side the membrane was a little pink in its posterior part. There was no mental

clouding; she resented being turned over for examination as it made her more giddy. She retched frequently. The optic disks were normal.

Mr. Sydney Scott kindly saw her with me, and the following points were noted: The patient heard a whisper at least 8 ft. from either ear; the appreciation of low tones was normal (sixteen vibrations per second being heard). Rinne's test was positive, and the upper tone limit normal (monochord 14·5 cm.). There was no pain in the ears and she had had none. The patient yawned frequently; knee-jerks were very brisk, Achilles jerks normal, plantar reflexes flexor; abdominal reflexes present and a well-marked *white* tache; there was slight impairment of movements of pronation and supination of the right forearm compared with the left. There was a *pointing error* in the vertical plane, the arm passed much too far upwards, especially on the right side; in the horizontal plane the finger fell below the starting point on both sides, but perhaps this was due to lack of effort on the part of the patient. Lumbar puncture showed the fluid to be under very decidedly increased pressure, and one and a half test-tubes were drawn off; the fluid was clear and normal. It was not considered wise to apply the caloric tests on account of the vertigo already present.

Dr. James Taylor also saw the patient. No definite diagnosis was arrived at, but it was agreed that the condition was due to some cerebellar irritation of unknown origin. Potassium bromide was given *per rectum*. The patient could only take small amounts of lemonade and peptonized milk for the next two or three days.

On March 24 objects still seemed to move from left to right. Vertical nystagmus was still present, combined with lateral nystagmus to the left when looking to the left. There was no vomiting.

March 27: Patient was much better; lateral nystagmus to the right.

March 28: Nystagmus was now right rotatory on looking to the right, but lateral when looking to the left; no vertigo; rhinitis considerable.

The patient continued to improve, and by April 12 could sit up, though this made her feel somewhat giddy; there was then slight vertical and right rotatory nystagmus. After that the patient made a perfect recovery and she is now back at work.

(*November* 17, 1916.)

Labyrinthine Irritation in a Patient on whom a Complete Mastoid Operation had been performed some years previously.

By Somerville Hastings, F.R.C.S.

M. J., a young woman, aged 31, had a left complete mastoid operation performed for chronic ear discharge eight years ago. The wound healed well, and for two years she had no trouble at all. Six years ago the discharge began again and was accompanied by tinnitus, and both have continued ever since. Three years ago she began to be troubled by giddiness, and this has also continued. The giddiness comes on at all times of the day and night, and she tends to fall to the left. When first seen on November 7, 1916, the left ear and mastoid cavity were filled by foul smelling epithelial debris and pus. On removal of this, ulceration was seen on the inner wall of the middle ear, immediately below the promontory. The hearing in the right ear was good, but nothing was heard on the left by air conduction. On the left mastoid C_{64} and C_{512} forks were heard when vibrating strongly. The fistula symptom was well marked, and increased pressure produced rotatory nystagmus to the right with falling to the left. The left labyrinth reacted readily to cold, and rotatory nystagmus to the right was produced in ten seconds.

I should be glad of the opinion of members as to whether a labyrinthectomy would be desirable should symptoms still persist now that the ear is being kept clean.

(*November* 17, 1916.)

A Doubtful Case of Labyrinthitis.

By E. D. D. Davis, F.R.C.S.

A married woman, aged 46, attended the hospital seven and half years ago, February, 1909, for left middle-ear suppuration of twelve months' duration, with a large perforation in the lower and posterior

part of the drum. She complained of tinnitus, which was arrested by compression of the carotid. She returned six weeks later with a mass of granulation tissue in the meatus, which suggested a malignant growth. The section merely showed granulation tissue, but a radical mastoid operation was performed by Major Waggett. The facial nerve was exposed and a temporary paralysis occurred, but the face recovered within two months, when the ear was healed and dry. After the operation the patient complained of attacks of vertigo, which have persisted up to the present.

When she was seen a fortnight ago the gait was staggering, and there was a tendency to fall to the left. There was no nystagmus, the pupils were equal and reacted to light; the disks were normal. The knee-jerks were exaggerated. Rombergism was well marked. Irrigation of the ear with lotion at 100° F. produced neither nystagmus nor giddiness. Irrigation with lotion at 105° F. for twenty seconds resulted in giddiness, with a tendency to fall to the same side, but there was no nystagmus. The mastoid area showed slight Eustachian discharge and debris. Hearing on the left side was considerably diminished, and bone conduction was reduced.

DISCUSSION.

Mr. J. F. O'Malley: In Mr. Hastings's case I advise the performance of labyrinthectomy: the patient seems much incapacitated by the attacks of giddiness and vertigo which occur in the most unexpected manner. At present the ear is useless as an organ of hearing. With regard to the tests, the disturbance of equilibrium points, in my opinion, to the labyrinth on that side being under-active. There is sufficient life in it to cause irritation, but the disturbance of equilibrium is associated with the over-action of the good side. There seems to be a difference between the fistula test response here and that which one usually gets in fistula of the external semicircular canal, which suggests a fistula, not in the canal, but somewhere round the stapes, and this may explain the altered direction of the nystagmus. I think this degree of labyrinthine defect may become dangerous at any moment by failing to indicate the spread of sepsis and, therefore, it would be well to perform labyrinthectomy.

Dr. H. J. Banks Davis: Is Mr. Somerville Hastings certain that the patient has not got cerebellar symptoms? She is in a miserable condition, and before so serious an operation is undertaken I think cerebellar abscess should be excluded.

Mr. Somerville Hastings (in reply): I thank members of the Section for the expression of their opinions. I thought of letting the case go a little

longer, and then, if the patient did not improve, of performing labyrinthectomy. I think it unlikely that she has a cerebellar abscess. The symptoms have been going on for three years, and have varied very little, which is not like the behaviour of a cerebellar abscess.

(*November* 17, 1916.)

Case of Malignant Disease of the Ear.

By SYDNEY SCOTT, M.C.

THE patient, H. R., aged 49, an engineer's fitter, had had malignant disease of the external ear which was first noticed eight and a quarter years ago.[1] Histologically the disease was a small spheroidal celled carcinoma like rodent ulcer, but contained cell-nests and involved lymphatic glands (Professor F. W. Andrewes). Three operations had now been performed during this period, and the whole of the left auricle with the bony meatus, the mastoid process, the parotid salivary gland, the whole of the sternomastoid muscle, and all lymphatic glands on the left side of the neck have been removed. The last operation was performed in May, 1916, when there was a small recurrence in the middle of the scar in the neck. Since this the left vocal cord has become paralysed, but there has been no sign of returning disease until the last few weeks, and now several nodules have appeared in the scar along the clavicle and in the posterior triangle. There is also a larger nodule which has commenced to ulcerate over the carotid sheath close to the angle of the jaw. An expression of opinion is sought as to whether any further attempts should be made to remove the disease.

The patient is presented in conformity with the recommendation of the Council of the Section that the after-history of cases shown in the past should be recorded.

DISCUSSION.

The PRESIDENT: I think it would be well to excise the outlying nodules, which are all quite small. It must be a chronic type of case. If the ulceration can be stopped, a great advantage will be gained. Are there any signs of secondary growths anywhere?

Mr. SCOTT: None, so far.

[1] The patient was first shown in January, 1914; see *Proc. Roy. Soc. Med.*, 1914, vii (Sect. Otol.), p. 23.

(*November* 17, 1916.)

A Post-mortem Specimen of a Temporal Bone from a Case of Cerebellar Abscess.

Shown by W. M. Mollison, M.C.

The left temporal bone from a patient who died with a cerebellar abscess which gave rise to no symptoms. The posterior surface of the petrous bone shows a small collection of pus lying in the pocket of dura mater over the meatus vestibuli.

(*November* 17, 1916.)

Anatomical Specimens of the Petrous Bone.

Exhibited by H. Banks Davis, M.B.

The specimens exhibited consist of many dissections of the temporal bone removed after death from patients who died from complications following suppurative otitis media, meningitis and cerebral abscess, and from patients who died of other diseases. Some of the dissections show clearly the relation of the facial nerve to the external semicircular canal.

In addition to these there are anatomical specimens of skulls and temporal bones which I have collected on the Continent and elsewhere for private and teaching purposes. They have been previously exhibited at International Congresses.

Section of Otology.

President—Captain H. J. Marriage, R.A.M.C.(T.), F.R.C.S.

(*February* 16, 1917.)

SPECIAL DISCUSSION ON WARFARE INJURIES AND NEUROSES.

Opening Address by the President.

The following remarks are based upon notes of cases kindly sent to me by Mr. Arthur Cheatle, Mr. E. D. D. Davis, Mr. Somerville Hastings, Mr. Pike and Dr. W. S. Syme, and also on an excellent paper published by Mr. Logan Turner, Mr. J. S. Fraser and Mr. W. J. Porter, a copy of which was sent to me by Mr. Fraser, in addition to the cases I have seen myself. I propose for the purpose of this discussion on warfare injuries and neuroses to deal with the subject under the following headings:—

(I) Injuries of the auricle and external meatus.

(II) Injuries of the middle ear.

(III) Injuries of the internal ear: (A) Direct injury to the labyrinth by a missile; (B) indirect injury of the labyrinth in fracture of the skull; (C) concussion deafness.

(IV) Psychical deafness.

(I) Injuries of the Auricle and External Meatus.

(*a*) Wounds of the auricle may be caused by a bullet passing through the ear or by small pieces of metal which either lodge in the auricle or pass through it; at times the ear is to a large extent torn away. In

dealing with these cases all that is usually required is to clean up the wound, remove fragments of metal, and apply antiseptic dressings. If the damage is very extensive it is often possible to save the greater part of the ear by performing a plastic operation, as the blood supply to the auricle is extremely good. The chief complication which is liable to occur is perichrondritis, with at times necrosis of the cartilage, and should this take place it is sometimes necessary to dissect out the cartilage before healing will take place.

(*b*) Wounds of the external meatus are usually caused by missiles which enter either in front of, or behind the ear, and pass right through the meatus; the cartilaginous meatus appears to be most frequently affected, though at times the bony canal may also be damaged. In its course the missile often injures the facial nerve and may also cause more or less injury to the mastoid process. In one case which I saw about a fortnight after the injury, a bullet entered just in front of the tragus, passed right through the external meatus, struck the tip of the mastoid causing a slight fracture which was seen by X-rays, and emerged behind and below the mastoid process. The wound of exit was about the size of a large hazel-nut and gradually filled up without any involvement of the mastoid. There was also some weakness of the lower part of the face, but this disappeared. In the meatus there were small perforations in the anterior and posterior walls, which were granulating; these were treated by syringing and plugging the meatus. The drum was not damaged, but there was some internal ear deafness probably due to concussion.

The following case of injury to the bony meatus is recorded by Mr. Arthur Cheatle: On July 1, 1916, a nearly spent machine gun bullet accurately entered the right meatus at right angles and without injuring the cartilaginous portion reached the bony meatus, passed through the anterior wall, was deflected downwards and backwards, and emerged behind the upper third of the sternomastoid on the same side, lodging in the collar badge. The man was knocked down, did not lose consciousness, but was dazed, giddy and sick. When seen a few days later, there was slight thin discharge from the ear, the cartilaginous meatus was normal, but a swelling in the anterior and inferior part of the deep meatus, from which projected a small granulation, obscured the anterior half of the membrane. A longitudinal, healing wound of exit, 1½ in. in length, was present behind the upper third of the sternomastoid on the same side. He complained of some deafness and tinnitus, with some difficulty and pain in moving his jaw at the

temporo-maxillary joint; no bony crepitus could be heard with the stethoscope on moving the jaw, but there was distinct synovial creaking. He could hear conversational voice at 6 ft., and the tuning-fork on the nose was referred to the injured side. There was no pain, giddiness, nystagmus nor facial paralysis; the nose, pharynx and nasopharynx were normal; the temperature was 99° F. The cartilaginous meatus was painted with iodine and lightly plugged with wool. After a few days the slight discharge became offensive and facial paralysis came on gradually. The ear was then syringed regularly with saline. In a few weeks the discharge ceased, and the facial paralysis gradually disappeared. It was then possible to examine the membrane which was seen to have been ruptured in the anterior inferior segment. The hearing became almost normal.

The interest of this case lies in the facts: (1) That a bullet should have so accurately entered the ear, and without causing any injury until it reached the deep meatus. (2) That so little immediate or lasting injury was inflicted. The bullet must have been a nearly spent one with a falling trajectory.

Cicatricial stenosis of the meatus very commonly results in these cases if there is extensive injury, and will cause some deafness, and in addition there may be some internal ear deafness.

Cheatle records the case of an antero-posterior bullet wound of meatus and auricle with labyrinthine (?) hæmorrhage and complete cicatricial closure of the meatus. A lieutenant, while looking through field glasses, was injured by a rifle bullet which, fired from a range of from 200 to 300 yards, passed through the palm of the left hand, glanced off the malar bone and passed through the meatus, cutting the auricle in half. He was unconscious for a few minutes and was giddy and sick on recovery. He had complete facial paralysis at first, but when examined by Cheatle two months after the injury, this had nearly disappeared. He said he had been quite deaf since the injury and that he had been unsteady in walking, as if drunk, until three weeks before examination. He also noticed that at first things tasted differently, but that had quite passed off. The wounds were healed fourteen days before examination, but with complete closure of the meatus. The deafness in the ear was absolute with Bárány's noise machine in the other, and rotation did not produce nystagmus. His own voice and the tuning-fork on the vertex and left mastoid were only heard on the right side.

(II) Injuries of the Middle Ear.

Rupture of Tympanic Membrane.—This has been very common and mainly due to high explosive, and is much more likely to occur when a bomb or shell bursts in a confined space such as a trench or dug-out. It is probably due to increased pressure on the drum, but one must not forget that with reference to windows the glass falls outwards owing to the rarefaction of the air after the explosion, and it is possible that this rarefaction may also have some effect on the drum. The perforation occurs in the membrana tensa, and among the cases submitted to me I have not found any record of injury to the membrana flaccida. From my own cases and from the notes of those sent to me, I could not determine that the rupture was liable to occur in any particular quadrant of the drum, but it seems to be most frequent in the lower half. Cheatle, in his notes, states that it was most frequently in the anterior and inferior quadrant; E. D. D. Davis states that it was always in the lower half; in my own cases there was a slight preponderance of ruptures in the posterior inferior quadrant, and Mr. Fraser in his article states that Japanese otologists have found that in 50 per cent. of cases the rupture is in the posterior part, in 24 per cent. in the anterior part, and in 26 per cent. along the handle of the malleus. In one case which I saw the drum had been torn from its attachment to the lower part of the tympanic ring, and Mr. Pike records a similar case. The size of the rupture varies enormously, from a small pin's head to the loss of half the drum. In cases seen within a few days of the injury, the edges are ragged and covered with dried blood, but later the edges become quite smooth.

In a case reported by Mr. Mollison at the last meeting of this Section, owing to an explosion at a munition factory the drum was completely destroyed and the incus blown into the antrum.

The condition of the nose and nasopharynx, as far as I could determine, had nothing whatever to do with the frequency of the injury, as in the majority of the cases examined by me both were quite healthy and free from deformity.

The commonest symptoms are marked deafness with, at times, loss of consciousness for a few minutes and often slight bleeding from the ear, which may be followed by a little serous discharge; rarely there may be tinnitus and slight vertigo due to involvement of the internal ear. As regards the hearing, I found that in most cases, when tested with tuning-fork C even a few days after the injury, the bone conduc-

tion was normal, but the air conduction was diminished, the amount of diminution varying according to the extent of the damage; in several cases the air conduction was also slightly subnormal when tested with tuning-fork C_5. Complete loss of hearing has not been observed in any of the reports which have come under my observation.

If no suppuration took place, I found healing occurred in from one to three months, and E. D. D. Davis gives the time as five to ten weeks, with almost complete recovery of hearing.

As regards the frequency with which infection of the middle ear occurred, I am not able to give any satisfactory statistics, but in the cases seen by me, about 30 per cent. had discharge when admitted to hospital over here. The majority of these healed quite well, with good recovery of hearing, and in only one case have I had to open up the mastoid, and that was in a patient who had extensive injury of the pinna and external meatus. Cheatle also states that he has only once had to operate on the mastoid, and I have received no notes of any other cases. The usual treatment for ruptured drums adopted over here has been to disinfect the meatus and to keep it plugged with gauze or wool, avoiding all syringing and drops.

Somerville Hastings reports an interesting case of hæmorrhage into the middle ear: On October 6, 1916, a man was wounded on the right cheek by a bomb. When examined on October 24, the handle of the malleus was injected, and behind the membrana tympani dark coloured blood was seen. The hearing was impaired, Politzer's acoumeter being heard at 2 in. Rinne's test was negative and the bone conduction normal. On October 30 bubbles were seen in the blood in the right middle ear and the hearing distance for the acoumeter was $2\frac{1}{2}$ ft. On November 13 all blood from the middle ear had been absorbed, the membrana tympani appeared a little retracted, and the acoumeter was heard at 3 ft.

Cheatle also reports a case of hæmorrhage into the middle ear due to parachute descent. An airman, aged 21 years 10 months fell a distance of 13,000 ft. in four minutes. The only ill-effects of the fall were a feeling of deafness and stuffiness in the left ear and a crackling when yawning. The left middle ear was found full of blood, with an intact membrane. The hearing was only slightly diminished. Nothing was done in the way of treatment, and in about a month the blood had become absorbed and the hearing had returned.

(III) Injuries of the Internal Ear.

(*A*) *Direct Injury of the Labyrinth by a Missile.*

These wounds as a rule are fatal, but in a few cases the patient has survived, and Cheatle has reported a particularly interesting one in which, on July 1, a rifle bullet entered the left temporal region above the zygoma and emerged just to the left of the second cervical vertebra. He was unconscious for about twelve hours, and was then able to crawl back. When seen over here on July 11 by Dr. Collier, his general condition was good, temperature 100° F., pulse 84. He was quite deaf in the left ear, from which there was a copious discharge of cerebrospinal fluid. He spoke with a husky voice. There was complete paralysis of the left trapezius and sternomastoid muscles, and of the left side of the soft palate. There was no facial paralysis. The pupils were equal and reacted to light. No loss of sensation. The tongue was protruded straight and the diaphragm moved well. A note on July 14 states he has had a variable temperature for some days, maximum 102° F.; some headache, dull and sleepy; herpes on lips. July 18: Temperature normal, pulse 64, respiration 18; general condition good; discharge of cerebrospinal fluid still profuse. August 1: All discharge from ear stopped; getting up. August 18: First examined by Mr. Cheatle, who made the following notes: "The deafness of the left ear is absolute. The tuning-fork and his own voice are referred to his right ear. Complete absence of vestibular reaction to cold syringing. No discharge from the ear; the membrane seemed to be replaced by cicatricial tissue, and no details could be made out. No Rombergism, no nystagmus. Recurrent laryngeal nerve paralysis of the left cord." Patient was discharged to a convalescent home with the wounds healed.

(*B*) *Indirect Injury of the Labyrinth in Fracture of the Skull.*

These may be caused by falls, blows on the head, or by bullet wounds. Fraser states in his paper[1] that: "If the force is applied at the side of the head in the direction of the petrous pyramid we get a longitudinal fracture which usually runs along the roof of the middle ear cleft . . . As a rule, in these cases the labyrinth capsule remains intact . . . On the other hand, if the force is applied at the back of the head, we get

[1] *Edin. Med. Journ.*, 1917, n. s., xviii, p. 112.

a transverse fracture which runs at right angles to the long axis of the petrous pyramid and always injures the labyrinth. As a rule, these fractures pass through the vestibule. The symptoms of such injuries are extreme or total deafness, marked disturbance of balancing, with loss of the cochlear and vestibular functions. If the labyrinth is injured there is an escape of cerebrospinal fluid along with blood. In the cases in which the labyrinth capsule is not injured, there is no escape of cerebrospinal fluid; but nevertheless deafness results from hæmorrhage into, or tearing of, the eighth nerve. . . . One or two of the cases with fracture of the labyrinth have been microscopically examined many years after the accident, and filling up of the hollow spaces of the labyrinth by new connective tissue and bone has been found, along with secondary degenerative atrophy of the nerve structures."

Cheatle quotes a case of bullet wound causing fracture of the base of the skull, with complete labyrinthine deafness on both sides. A bullet entered in front of the right tragus and emerged at a closely corresponding point on the left side. The anterior meatal wall was evidently hit by the bullet on the right side and it just missed the left, for the right ear was discharging and the meatus was filled with granulations. Complete bilateral labyrinthine deafness resulted. Patient had been desperately ill with left hemiplegia and discharge from the nose and wound of exit. Hæmorrhage from the nasopharynx had necessitated ligature of the right common carotid artery. The X-ray picture appeared to show a piece of metal at the base of the brain, behind the sella turcica, with radiating fracture to the vault.

(*C*) *Concussion Deafness.*

This may be due to the constant noise of the guns firing day after day, or to the explosion in the immediate neighbourhood of the patient of a shell containing high explosive. In the former case, the results are similar to those seen in peace time in naval gunnery officers and boiler-makers, and in my experience a slight amount of permanent deafness usually persists if the patients have been frequently exposed to the noise for long periods. The deafness due to the explosion of a shell is generally for a short time very severe, and at times the patient has been rendered unconscious, but as far as I know the deafness has never been absolute.

As regards symptoms the patient has often only complained of intense deafness with headache, but at other times there has been marked

tinnitus in addition, and in the more severe cases there may be nausea and vomiting; it is more common for only one ear to be affected, though both may be, especially if the shell bursts just in front or behind him. When tested with tuning-fork C, I have found a positive Rinne with marked diminution of both air and bone conduction, and with tuning-fork C_5 also marked diminution of the air conduction. The drums have been normal.

A large proportion of these patients recover their hearing in the course of one or two months, but others improve very little. In one case which I saw in 1914 a shell burst just behind a lieutenant without causing him any wound, but rendered him completely unconscious for about an hour, during which time the Germans passed over him and relieved him of anything of value. When he came to, he noticed marked deafness of both ears, with intense headache, but he had no hæmorrhage from either ear, no discharge, no tinnitus and no vertigo. I saw him four days later, when he heard spoken words on each side at a distance of 2 ft., but my watch not at all, though it can usually be heard at $3\frac{1}{2}$ to 4 ft. With tuning-fork C air conduction and bone conduction were both much subnormal, the air conduction being better than the bone conduction, and with tuning-fork C_5 the air conduction was again much subnormal. Both drums were healthy. Throat and nose healthy. No nystagmus. The patient steadily improved and eighteen days after the explosion the hearing on both sides had become normal.

The treatment I have adopted in these cases is complete rest in bed with the administration of bromides in the early stages and strychnine later.

As regards prognosis, I know no means of telling whether the hearing will recover or not, but as far as I can judge from cases I have seen, if marked improvement does not occur within six to eight weeks, the outlook is distinctly bad. Even where the hearing returns, tinnitus often persists. The exact lesion in these cases I have been unable to determine, as I have not been able to find any record of a post-mortem examination, but it is probably chiefly peripheral, as it is rare to find marked concussion deafness associated with a ruptured drum, suggesting that the rupture of the drum lessens the force of the explosion against the internal ear; it is possible, however, that in some cases at any rate some central injury may also occur, such as hæmorrhages into the pons, medulla or cerebellum. I hope some of the members present to-night may be able to clear up this point.

(IV) Psychical Deafness.

This is usually bilateral, and is commonly seen in those who have suffered a severe shock—e.g., by being buried by a shell, without any visible injury being present. The deafness is usually absolute, nothing being heard either by air or bone conduction. It is generally accompanied by other nervous signs and symptoms, such as loss of voice, narrowing of the field of vision, tremors, irregular paralyses and areas of anæsthesia. Spontaneous nystagmus is absent. Milligan and Westmacott, in their paper read before the Laryngological Section in May, 1915, attribute this to "a temporary suspension of neuron impulses from the higher cortical cells of the central nervous system to the periphery." They state: "Our view is that the hiatus or synapse interfering with the flow of nervous stimuli is a central and not a peripheral one, for the reason that in so many of the cases of sudden blindness and sudden deafness, no trace of any peripheral organic lesion was demonstrable, and, moreover, the rapid recovery of so many of the patients we have observed is a strong argument that no lesion was ever present." To my mind this is a reasonable explanation, but I shall be glad to hear the views of others, especially of any neurologists who happen to be present.

Cheatle has recorded the two following cases: (1) During a bombardment a private suddenly became unconscious without any definite cause and remained so for two days. On recovering consciousness complete bilateral deafness was at once noticed; no giddiness, no bleeding of the ears; had spasms of the neck. No organic lesion was discovered, but the deafness remained, and he could not hear the loudest shouting or his own voice. The vestibular reaction to cold syringing was normal. (2) A Belgian soldier was blown off his bicycle in the retreat from Antwerp, and had been completely deaf in both ears, aphonic and paralysed in his left arm and leg from that time until July, 1916. Rapid recovery occurred on the application of the battery after resistance to many forms of treatment, including hypnosis.

These cases often recover with a counter-shock, with rest and massage, or upon giving an anæsthetic, and at times the administration of asafœtida and valerian is useful.

To distinguish this form of deafness from malingering is sometimes difficult, but it can generally be accomplished if a thorough examination is made, as the malingerer is very liable to give himself away during the performance of the various tests. The malingerer is usually sullen

and defiant, has all his wits about him, and replies after deliberation, whereas a patient with psychical deafness has signs and symptoms of nervous breakdown.

Just one word about obturators. I believe they are of distinct benefit in saving the hearing, and that one of the simplest and best forms is plasticine wrapped in gauze; this can be moulded to the shape of the external meatus and is easily put in and withdrawn. An obturator largely advertised is the Mallock Armstrong, which is made in several sizes, but to my mind it is not so comfortable as plasticine. Plugs made of celluloid should never be used, as I am told that on one ship during a battle several men had their ears damaged by the flash of a shell setting fire to the celluloid plugs which they wore.

In conclusion, Gentlemen, I regret that these remarks are very incomplete owing to the comparatively small number of cases which came under my notice, but I hope they will suffice to start a good discussion.

The Morbid Anatomy of War Injuries of the Ear.

By J. S. Fraser, M.B., F.R.C.S.E., and John Fraser, M.D., F.R.C.S.E., Captain R.A.M.C.

(From the Laboratory of the Royal College of Physicians, Edinburgh.)

Introduction.

From the point of view of morbid anatomy, war injuries of the ear may be classified as follows:—

(1) *Direct injuries* due to bullets or pieces of shrapnel or high explosive shell. (2) *Indirect injury* due to blows or falls on the head. These injuries may be subdivided into (*a*) those without fracture of the labyrinth capsule, and (*b*) those with fracture of the labyrinth capsule. (3) *Noise deafness* due to prolonged or intense gun fire, and (4) "*shell*" *or* "*explosion*" *deafness* (labyrinth concussion).

(1) *Direct Injuries.*—Fracture of the mastoid process or of the external meatus may occur with or without splintering of the bone. The tympanum and labyrinth may also be involved in these cases. Further, the middle and inner ear may be injured by bullets entering through the facial bones and emerging through the mastoid or remaining embedded in the temporal bone. If the patient lives the nature of the injury can best be ascertained by means of good

radiograms—both lateral and antero-posterior. In severe and fatal cases of comminuted fracture of the petrous bone microscopic examination of the ear would be a matter of extreme difficulty, but where the injury is less severe this method may be of use. In many of these cases of direct injury suppurative otitis media occurs as a result of infection, either carried in by the foreign body at the time of the injury or due to secondary infection through the Eustachian tube or external meatus. By the kindness of Lieutenant-Colonel Caird, R.A.M.C., we are able to record a case in which infection took place (Case I, *see* later).

(2) *Indirect injuries* of the ear due to fracture of the base of the skull in civil life were recently dealt with by one of us (J. S. Fraser) before this Section. We have recently examined the middle and inner ear from a case of bullet wound of the fronto-parietal region attended by laceration of the brain and hæmorrhage into the subarachnoid space (Case II, *see* later).

(3) *Noise deafness* is due to physiological over-stimulation of the auditory apparatus. Recent researches have shown that (i) *air conduction of sound is of paramount importance* in the production of noise deafness. Conduction through the tissues of the body, including the cranial bones, is of little account. (ii) *The neuro-epithelium (hair cells) of Corti's organ are first affected*, later the supporting cells are involved. The ganglion cells and nerve fibres are secondarily affected. The condition is one of so-called "degenerative neuritis." (iii) *The part of Corti's organ affected depends on the pitch of the sound.* If the noise be of high pitch the neuro-epithelium at the base of the cochlea is involved. If the noise be of medium pitch, Corti's organ in the middle coil is affected; while if the noise be of low pitch, degeneration is found in a portion of Corti's organ nearer the apex of the cochlea. These experiments confirm Helmholtz's theory of the peripheral analysis of sound.

It is very difficult or impossible to draw a line between cases of "noise" deafness and those of "shell" or "explosion" deafness, as both conditions are probably due to excessive movements of the atmosphere conveyed to the labyrinth. The explosion of a shell not only causes a great mass movement of air but produces a loud noise. It is stated, however, that the "blow" produced by the condensation of air following the explosion, reaches the ear before the noise vibration, and that it may drive the stapes inward and fix it in the oval window so as to lessen the bad effect of the loud noise which follows. In somewhat the same way the stapes is fixed in Gellé's experiment.

(4) *"Shell" or "Explosion" Deafness (Labyrinth Concussion).*—According to Lermoyez "shell" deafness is the true war deafness. Various theories have been put forward as to the pathology of this condition. (*a*) In many cases we are actually able to observe a *rupture of the tympanic membrane*, accompanied by a certain amount of *hæmorrhage*. It has been stated that in cases of shell explosion in which the drumhead ruptures, there is less likelihood of damage to the delicate structures of the membranous labyrinth than in cases in which the tympanic membrane does not give way. In the same way the internal mechanism of a watch, which has been dropped on the ground, is more likely to escape injury if the fall results in the fracture of the watch glass. Mere rupture of the drumhead, however, unless accompanied by some lesion in the labyrinth, auditory nerve or brain, would only produce a slight diminution of hearing. We know, however, that patients suffering from severe shell deafness exhibit marked or total loss of hearing. Some further lesion in the auditory apparatus must therefore be sought for.

(*b*) It has been stated that *hæmorrhages occur in the peri- or endolymphatic spaces* of the inner ear and that the delicate neuro-epithelial sacs and tubes of the *membranous labyrinth are ruptured* by the violent concussion caused by the explosion. It is supposed that in this way a gross mechanical effect is produced in the inner ear. It would appear, however, that the structures of the membranous labyrinth are well protected from concussion because they are suspended in a lymph bath inside the hollow spaces of the bony labyrinth.

Hæmorrhage may also occur in the *internal auditory meatus* with or without *rupture of the nerve fibres* which pass from the fundus of the meatus through rigid bony canals to the cochlea, utricle, saccule and cristæ of the canals.

(*c*) It has been suggested that apart from these gross mechanical changes ((*a*) and (*b*)) the explosion and the loud noise may *destroy the delicate nerve endings in the cochlea*, and so result in paralysis. The loud sound due to the explosion may paralyse the hair cells of Corti's organ, somewhat in the same way as the nerve structures of the macula in the retina are paralysed by the rays of the sun in "eclipse" blindness. According to this theory "shell" deafness, like "noise" deafness, is due to paresis or paralysis following over-stimulation. Some observers hold that the change is a biochemical one, while others believe that it is of a molecular nature. Theodore has microscopically examined one case of labyrinth concussion followed by total deafness, and found a condition

of degenerative neuritis similar to that described by Manasse and Wittmaack in old people.

(*d*) It has been stated that in cases of "shell" deafness the lesions are probably to be found in the brain—e.g., hæmorrhages in the pons, medulla, and cerebellum, involving the central connexions of the auditory and vestibular nerves. Milligan and Westmacott have suggested that shell deafness is due to a temporary interference with the neuron connexions in the higher brain centres. They believe that the abrogation of function is *not* due to an organic lesion.

Present Investigation.

The writers have examined the middle and inner ears from four cases of "shell" or "explosion" deafness. In each case the temporal bone on the injured side was removed by one of us (J. Fraser) within four or five hours of death. The labyrinth was not opened, but the specimen was at once wrapped in gauze soaked in 5 per cent. formol, then in waterproof tissue, and was finally surrounded by cotton-wool and packed in a cigarette tin. The lid of the tin was fixed on with adhesive plaster. All the specimens arrived in good condition, and were at once prepared for microscopic examination according to the method described by one of us (J. S. Fraser) in the *Journal of Laryngology, Rhinology and Otology* (December, 1913, page 638).

It is doubtful if our methods are delicate and accurate enough to discover fine changes in the neuro-epithelium of the labyrinth due to shell or bomb explosion, especially in cases like the present ones (Nos. III, IV, V and VI), where the hollow spaces of the labyrinth were not opened soon enough after death to allow the entrance of the fixing fluid. Further, the specimens had a prolonged journey from the Front to their destination in Edinburgh. Under these circumstances it would not be right to be dogmatic about fine changes in the neuro-epithelium or ganglion cells of the labyrinth. In one or two cases the writers have found displacement of the cupulæ of the canals and of the otolith membrane of the utricle and saccule. These may, or may not, be artefacts. In order to be accepted they would require confirmation by other observers.

There can, of course, be no doubt about such changes as rupture of the drumhead or extensive hæmorrhages into the middle-ear spaces or the internal meatus. It is interesting to note that in this latter position the hæmorrhages occur at the point where the nerves, after passing

to the fundus of the meatus, enter the fine bony canals. It is just at these parts that one would expect the small vessels to rupture and the nerve fibres to give way. It appears probable that the examination of the inner ear from cases of "shell" deafness, which die from other causes some months or even years after the shell explosion, would afford information of great value as to the true pathology of the condition. The microscopic changes in the membranous labyrinth and eighth nerve would be much more evident at this period. It is therefore very important that every opportunity of examining the inner ear in such cases should be taken advantage of. We are of opinion that the sudden severe explosion and the loud sound accompanying it may probably paralyse the neuro-epithelium of the labyrinth in a manner analogous to—but more severe than—that in which noise deafness is brought about in boiler makers. If the observations of foreign otologists regarding "noise" deafness be applicable to cases of "shell" deafness, one would expect that "degenerative neuritis" would occur—at least in severe cases—and that certain definite changes would be found in the neuro-epithelium of the labyrinth and in the auditory and vestibular ganglia and nerves of soldiers who had suffered from severe shell explosion with its consequent deafness, tinnitus, giddiness and loss of balancing.

Our findings lend little support to the view that in "explosion" or "shell" deafness (1) large hæmorrhages occur in the peri- or endolymphatic spaces of the labyrinth, or (2) that rupture of the delicate neuro-epithelial sacs and tubes of the membranous labyrinth takes place. The changes we have observed will be found in detail in the reports of the microscopical examination of four cases of "shell" or "explosion" deafness (Cases III, IV, V, and VI). Briefly summarized these changes are as follows: (*a*) Rupture of the drumhead in Cases III, IV and VI. (In Case V a large plug of wax was present in the external meatus and probably for this reason the tympanic membrane was not injured.) (*b*) Hæmorrhage in the middle-ear spaces in all four cases. (*c*) Slight hæmorrhage in the scala tympani in the region of the round window and basal coil of the cochlea (Case IV). (*d*) Slight degenerative neuritis of the cochlear apparatus?—e.g., loss of hair cells, slight flattening of the acoustic papilla and disappearance of some of the supporting cells, especially in Case IV; shrinkage of the ganglion cells of the spiral ganglion; uneven staining of the cochlear nerve. (All these may be post-mortem changes.) (*e*) Displacement of the otolith membrane of the saccule or utricle (Cases IV and V), artefacts? (*f*) Dis-

placement of the cupula of one or more of the canals (Cases IV and V) (It is quite probable that (*e*) and (*f*) are artefacts.) (*g*) The vestibular apparatus, as was to be expected, showed less change than the cochlear. The neuro-epithelium of the saccule and utricle and the cells of the vestibular ganglion in the internal meatus appear to be better preserved than the corresponding parts of the cochlear apparatus. (*h*) Hæmorrhage was observed in the fundus of the internal meatus at the points where the nerves enter the bony canals (Cases III, IV, and V).

CASE I.—DIRECT INJURY BY SHRAPNEL.

The patient presented a shrapnel entrance wound in front of the left tragus. There was no exit wound. Acute suppurative otitis media supervened and the patient suffered from discharge from the left ear and severe deafness. The case was only admitted to Colonel Caird's ward in the Royal Infirmary, Edinburgh, about a month after the injury. On admission (April 10, 1916) the pulse was 72 and the respirations 20 per minute. Temperature 97° F.

On April 16 there was a sudden rise of temperature to 103·6° F., and the pulse rose to 104. The patient complained of pain in the left ear and there was an increase in the aural discharge. On examination granulations were seen on the anterior wall of the left external meatus.

April 18, 1916: Temperature 101° F. Frontal headache present and pain in the right parietal region. Right ear normal.

April 19, 1916: Temperature 97° F., pulse 66. Occipital pain present with nausea and vomiting. The patient was examined by Dr. Logan Turner, who found slight facial paresis on the left side; spontaneous nystagmus to the left with a pointing error at the left wrist and shoulder joints; pain present on pressure over the neck muscles; mastoid tenderness on the left side and complete deafness in the left ear. The tuning-fork on the vertex was lateralized to the right (good) ear! The patient was too ill for the caloric test. On lumbar puncture the cerebrospinal fluid was found to be clear and not under increased tension.

Operation (April 19, 1916) by Colonel Caird. Granulation tissue found in the antrum along with some particles of nickel. The cerebellum was exposed behind the sinus and was found not to pulsate. On exploration a teaspoonful of pus was evacuated (*Staphylococcus albus* on culture). The cerebellar abscess was drained.

April 20, 1916: Temperature and pulse normal, headache less severe.

April 21, 1916: Temperature rose to 102° F. at 5 p.m. The patient showed signs of distress and died immediately.

Post Mortem.—Cerebellar abscess with early meningitis.

Macroscopic examination of right ear: Pus in the external meatus; small perforation of tympanic membrane; the Eustachian tube contains blood-stained mucus; granulations in the mastoid antrum; saccus endolymphaticus

healthy; sigmoid sinus and jugular bulb thrombosed along with the inferior petrosal sinus. (It will thus be seen that three intracranial complications were present.)

Microscopic Examination of Right Ear.

External Meatus.—Lining membrane swollen and congested.

Tympanic Membrane.—Upper part of drumhead very thick and infiltrated; lower part perforated. There is a small irregular piece of bone with granula-

FIG. 1. (Case I.)

Direct injury of ear by shrapnel, followed by suppurative otitis media, cerebellar abscess, &c. Horizontal section 140, × 11 diam. (hæmatoxylin and eosin). 1, head of malleus dislocated from 5, body of incus; 2, facial nerve; 3, ampullary end of superior canal with coagulated lymph; 4, lateral canal.

tions around it in the middle of the drumhead. The tympanic membrane in its lower part is adherent to the inner wall of the tympanic cavity.

Tympanum and Middle-ear Spaces.—The mucous membrane of the air cells in the roof of the antrum is swollen, congested and infiltrated with small cells.

Muco-pus is present in the cells and the bony walls are eroded in parts. At other parts there is some new bone formation in the walls of the cells. Some of the air cells contain a little blood. There is marked erosion of the bone on the inner wall of the attic. The mucosa of the tympanic cavity is swollen and polypoid. The canal for the tensor tympani is eroded. There is a fracture of the neck of the malleus—the lower part of the neck along with the handle being drawn inwards by the tensor tympani. The squamous epithelium of the

FIG. 2. (Case I.)

Direct injury of ear by shrapnel, followed by suppurative otitis media, cerebellar abscess, &c. Horizontal section 210, × 13 diam. Shows upper part of oval window with vestibule. 1, swollen mucosa in niche of oval window; 2, foot-plate of stapes; 3, meningitis in internal meatus; 4, neuro-epithelium of utricle with 5, detached otolith membranes; 6, facial canal (the nerve is absent—artefact).

drumhead is still attached to this lower fragment. The head of the malleus is healthy as is also the body of the incus, but the two bones are widely separated and the joint space is filled with granulation tissue. The foot-plate of the

stapes is fractured (*see* fig. 3, No. 5); the annular ligament shows evidence of early invasion by the inflammatory process in the middle ear. The stapedius muscle is normal, as is also the joint between the incus and stapes. The membrane of the round window is thickened and the niche is full of pus. The Eustachian tube is full of muco-pus and its lining membrane is swollen.

Labyrinth Capsule.—The vessels of the fossa subarcuata are congested. There is a small hæmorrhage in the facial canal but the nerve is normal.

FIG. 3. (Case 1.)

Direct injury of ear by shrapnel, followed by suppurative otitis media, cerebellar abscess, &c. Section 240, × 17 diam. (hæmatoxylin and eosin). Shows 1, rupture of drumhead; 2, middle portion of drumhead greatly swollen and infiltrated; 3, malleus in tympanic cavity; 4, erosion of incus; 5, fracture of stapes; 6, facial canal empty (artefact); 7, chorda tympani; 8, posterior part of drumhead.

Labyrinth Contents: (1) *Cochlea.*—There is hæmorrhage in the scala tympani of the basal and middle coils. Corti's organ presents its normal mound-like appearance in the middle coil, and the pillar cells are clearly seen.

The cochlear ganglion appears to be healthy. There is hæmorrhage in the cochlear opening of the perilymphatic aqueduct.

(2) *Vestibule.*—The neuro-epithelium of the saccule and utricle appear to be desquamated and the otolith membrane is separated. There is a small hæmorrhage in the perilymphatic space in the neighbourhood of the vestibular opening of the endolymphatic aqueduct; elsewhere the aqueduct is normal. (It

FIG. 4. (Case I.)

Direct injury of ear by shrapnel, followed by suppurative otitis media, cerebellar abscess, &c. Section 265, × 13 diam. (hæmatoxylin and eosin). 1, greatly swollen drumhead; 2, handle of malleus separated from drumhead and lying in tympanic cavity; 3, squamous epithelium of drumhead attached to malleus; 4, blood in scala tympani of cochlea; 5, infiltrated posterior part of annular ligament (early stage of invasion of vestibule); 6, inco-stapedial joint, normal.

must be remembered that invasion of the vestibule through the oval window was just beginning.)

(3) *Canals.*—The superior canal shows desquamation of the neuro-epithelium of the crista. Coagulated lymph is present but the cupula is absent. The external canal is normal. The posterior canal shows some pus cells in the perilymph space, and hæmorrhage is also present at the ampullary end. The neuro-epithelium of the crista of the posterior canal is desquamated.

FIG. 5. (Case I.)

Direct injury of ear by shrapnel, followed by suppurative otitis media, &c. Section 360, × 11 diam. (hæmatoxylin and eosin). Shows, 1, swollen, engorged, and infiltrated mucosa on promontory; 2, meningitis in internal meatus; 3, hæmorrhage in scala tympani of basal coil; 4, hæmorrhage in perilymph space of posterior canal; 5, fracture of bone between round window niche and crista of the posterior canal; 6, niche of round window.

Internal Meatus and Nerves.—Meningitis is present within the arachnoid sheath, but the eighth nerve has been torn out at the post-mortem.

Summary.

The case was one of shrapnel injury of the left ear followed by suppurative otitis media. When the patient was admitted to the Royal Infirmary, Edinburgh, about a month after the injury, symptoms of cerebellar abscess were present. Operation revealed mastoiditis and small metallic particles in the mastoid antrum. In spite of the evacuation of the cerebellar abscess the patient died. The autopsy showed early meningitis and sinus thrombosis in addition

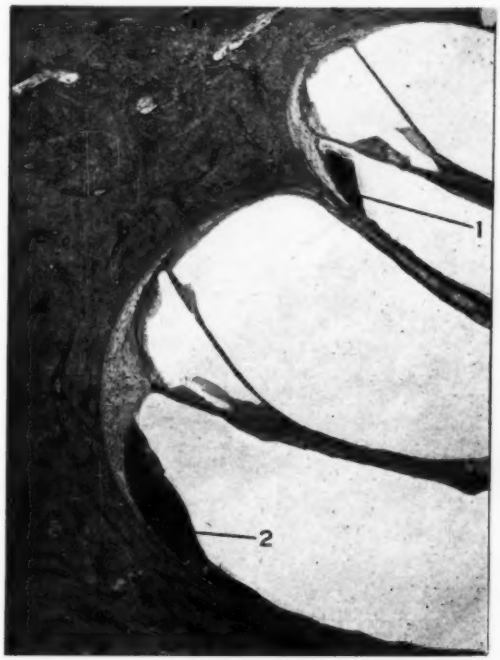

FIG. 6. (Case I.)

Direct injury of ear by shrapnel, followed by suppurative otitis media, cerebellar abscess, &c. Horizontal section 240, × 28 diam. (hæmatoxylin and eosin). Shows, 1 and 2, hæmorrhage in scala tympani in middle and basal coils.

to the cerebellar abscess. Microscopic examination of the ear demonstrated a thickened, infiltrated and perforated drumhead with fracture of the malleus. The lower part of the handle was separated from the drumhead and drawn inward by the tensor tympani. (We are unaware of any previous record

of a similar condition.) The mucosa of the middle ear was distinctly swollen, and the tympanic cavity, antrum and cells were full of pus. The joint between the head of the malleus and the body of the incus was dislocated. The inco-stapedial joint was healthy, but there was early invasion of the vestibule through the annular ligament. The cochlea showed hæmorrhage in the scala tympani of the basal and middle coil and in the opening of the perilymphatic aqueduct, but the nerve apparatus of the cochlea appeared almost

FIG. 7. (Case I.)

Direct injury of ear by shrapnel, followed by suppurative otitis media, cerebellar abscess, &c. Horizontal section 270, × 38 diam. (iron hæmatoxylin). Shows the normal (?) condition of Corti's organ in apical and middle coils. The nerves in the bony spiral lamina stain well.

healthy. The neuro-epithelium of the saccule and utricle and of the cristæ of the canals was desquamating. The internal meatus showed meningitis.

Note.—Several of the changes noted in this case may have been due to small pieces of shrapnel casing—e.g., fracture of the malleus and stapes, hæmorrhage in the scala tympani and fracture of bony capsule of ampulla of posterior canal.

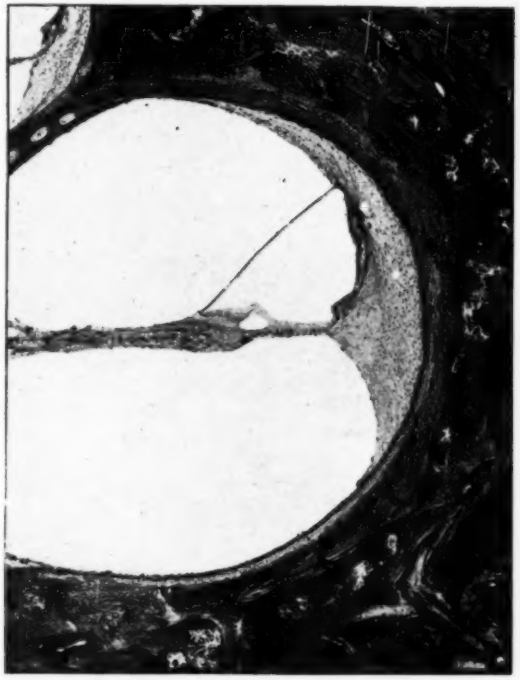

FIG. 8. (Case I.)

Direct injury of ear by shrapnel, followed by suppurative otitis media, cerebellar abscess, &c. Horizontal section 270, × 38 diam. (iron-hæmatoxylin). Shows condition of Corti's organ in upper part of basal coil. The acoustic papilla is reduced to a layer of flattened cells. The stria vascularis is normal.

CASE II.—INDIRECT INJURY OF THE EAR, DUE TO BULLET WOUND.

The patient was hit in the fronto-parietal region and the bullet came out just above the ear on the same side. There was no apparent injury of the ear itself, but the parietal bone was shattered and the brain lacerated.

Microscopic Examination.

The external meatus and the tympanic membrane are normal.

Middle-ear Spaces.—Free blood is present in the lower part of the tympanic cavity, in the niches of the round and oval windows, and in the mastoid antrum.

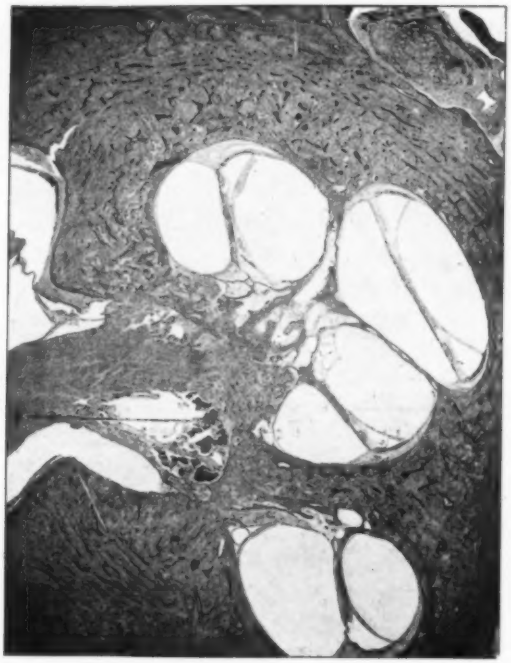

FIG. 9. (Case II.)

Bullet wound of fronto-parietal region, followed by intracranial hæmorrhage no direct injury of ear. Vertical section 60, × 11 diam. (hæmatoxylin and eosin). Shows, 1, hæmorrhage in fundus of internal meatus between fibres of cochlear nerve. Corti's organ appears normal in middle and apical coils.

Labyrinth Contents.—The nervous structures of the inner ear appear to be normal, but a little blood is present in the aqueduct of the cochlea, near its cranial end, and also around the vestibular nerve to the utricle.

Internal Meatus.—There is a considerable amount of hæmorrhage within the arachnoid sheath which surrounds the seventh and eighth nerves. The

bleeding is specially marked in the fundus of the meatus and extends along the facial nerve to the geniculate ganglion. This hæmorrhage appears to have been due to the injury to the brain and meninges.

(A similar case was recorded by Mr. Sydney Scott in the *Proceedings of the Royal Society of Medicine*, 1915, Otological Section.)

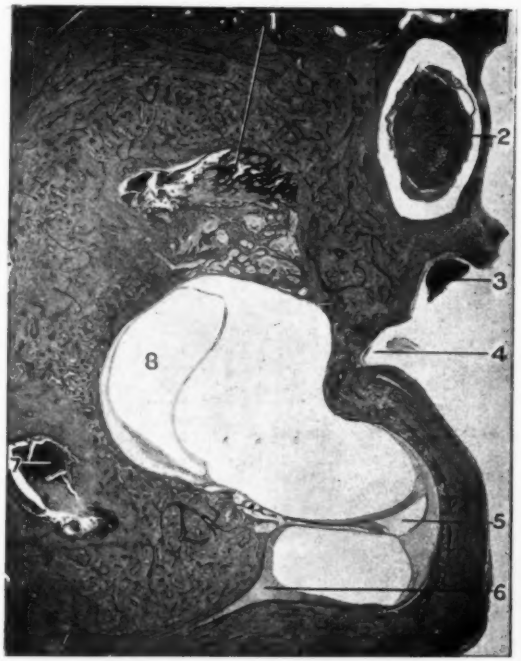

FIG. 10. (Case II.)

Bullet wound of fronto-parietal region, followed by intracranial hæmorrhage. No direct injury of ear. Vertical section 185 (hæmatoxylin and eosin). Shows 1, hæmorrhage along facial nerve above vestibule; 2, facial nerve on inner wall of tympanum; 3, hæmorrhage; 4, niche of oval window; 5, intravestibular part of cochlea; 6, opening of perilymph aqueduct; 7, hæmorrhage along nerve to ampulla of posterior vertical canal.

CASE III.—SHELL DEAFNESS.

This soldier was taking cover in a house when a high-explosive shell hit the house and burst in the room in which he was. The patient was wounded in many places. On admission to the Casualty Clearing Station the patient

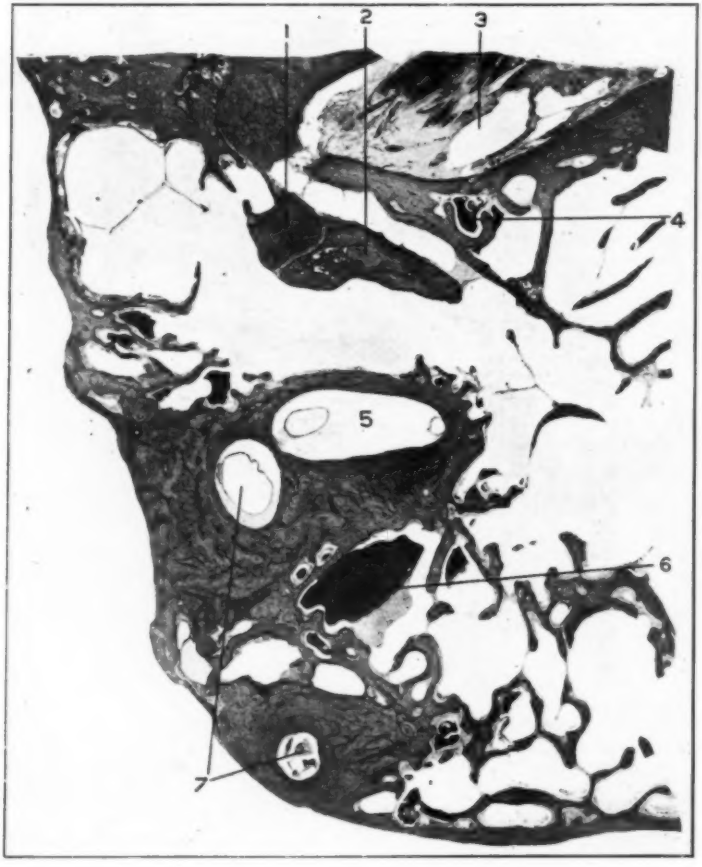

FIG. 11. (Case III.)

High-explosive shell injury of ear. Horizontal section, No. 60, × 6 diam. Shows hæmorrhage in border cells and also in air cells on inner wall of antrum. 1, malleus; 2, incus; 3, external meatus; 4, blood in "border" cell; 5, lateral canal; 6, blood in air cell; 7, two ends of superior vertical canal [the nearer (smooth) end contains bone chips—artefact].

was found to be semiconscious with subnormal temperature, small rapid pulse, cold clammy skin and multiple small wounds. In fact he showed the usual signs of high-explosive shell injury, including marked deafness. He died twenty-four hours later from shock.

Naked-eye examination showed a large ragged hole in the anterior part of the left tympanic membrane. The inner wall of the tympanic cavity was seen to be congested. The mastoid antrum and air cells appeared to be normal along with the Eustachian tube, saccus, jugular bulb and carotid canal.

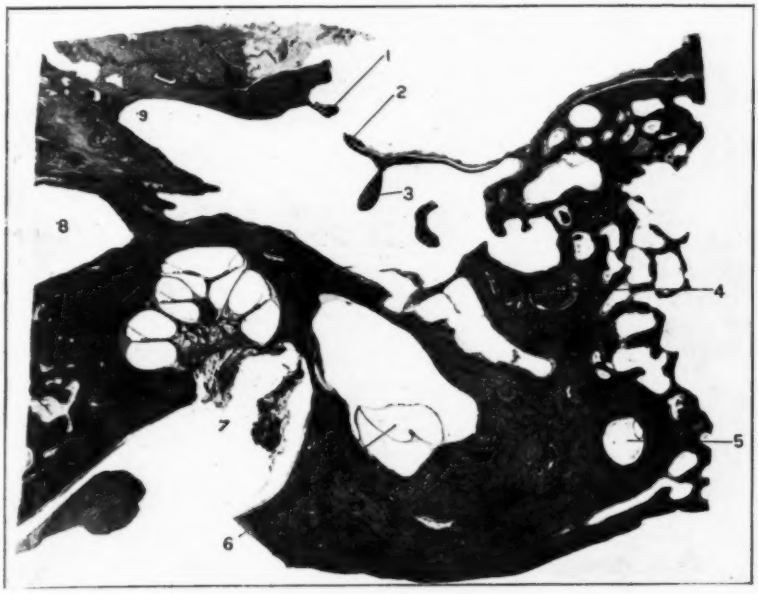

FIG. 12. (Case III.)

High-explosive shell injury of ear. Horizontal section, No. 200, × 6 diam. (hæmatoxylin and eosin). Shows rupture of anterior part of drumhead with everted edges. 1, anterior portion of ruptured drumhead (everted); 2, posterior margin of rupture, also everted; 3, malleus; 4, facial nerve with stapedius to the left; 5, smooth end of posterior vertical canal; 6, lower part of utricle with crista quarta; 7, internal meatus; 8, carotid canal; 9, tubal part of tympanum.

Microscopic Examination.

Drumhead.—This is ruptured in the anterior inferior part. The edges of the rupture are everted and show a little hæmorrhage. The rupture extends as far as the handle of the malleus.

Middle-ear Spaces.—There is a little hæmorrhage in the floor of the tube, in the niche of the round window and also on the promontory. There is slight bleeding in the air cells in the roof of the mastoid antrum and also in the border cells between the aditus and the external meatus. The tympanic ossicles and muscles are healthy. The oval window and the facial nerve are normal.

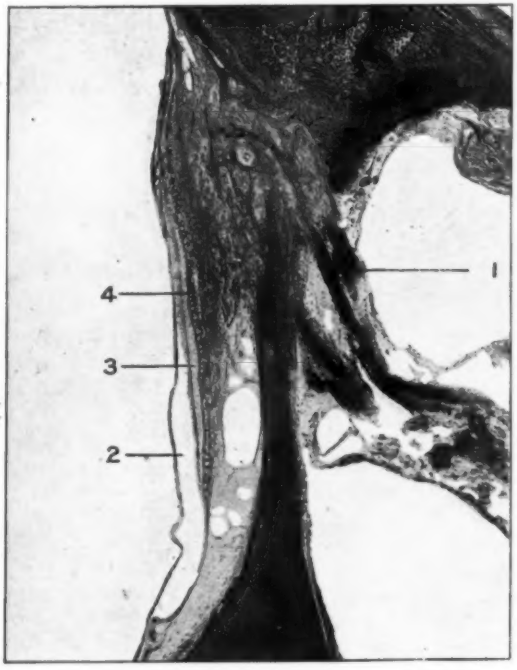

FIG. 13. (Case III.)

High-explosive shell injury of the ear. Horizontal section, No. 203, × 33 diam. (iron-hæmatoxylin). Shows normal condition of sacculus. 1, nerve to saccule; 2, cavity of saccule; 3, otolith membrane; 4, neuro-epithelium of saccule.

Hollow Spaces of the Labyrinth: (1) *Cochlea.*—The scala tympani and the scala vestibuli are healthy. Corti's organ is well formed; the pillar cells and other supporting cells are well seen, but the hair cells are not visible. The nerves in the modiolus and the spiral ganglion are normal.

(2) *Vestibule.*—The utricle, saccule and endolymphatic duct, along with the perilymph spaces all appear to be healthy.

(3) *Canals.*—These appear to be normal, along with the nerves supplying the cristæ.

Internal Meatus and Nerves.—The vestibular ganglion is healthy, but there are small hæmorrhages in the fundus of the internal meatus where the branches of the cochlear nerve to the basal coil enter the modiolus, as well as in the narrow canal for the nerve to the ampulla of the posterior canal.

(*See* figs. 11 to 17.)

FIG. 14. (Case III.)

High-explosive shell injury of the ear. Horizontal section, No. 203, × 48 diam. (iron-hæmatoxylin). Shows hæmorrhage in vestibular nerve and normal condition of ganglion in the internal auditory meatus. 1 and 3, hæmorrhage between fibres of vestibular nerve; 2, vestibular ganglion (normal).

Summary.

This case of high-explosive shell injury of the ear shows certain definite changes—viz., rupture of the tympanic membrane, slight hæmorrhage in the middle-ear spaces and in the fundus of the internal auditory meatus. The structures of the membranous labyrinth show normal conditions.

CASE IV.—INJURY TO THE EAR DUE TO THE BURSTING OF A RIFLE GRENADE.

The grenade burst close to the left side of this soldier and caused extensive general injury. (Rifle grenades, though much smaller than shells, contain a very powerful form of high explosive.—J. Fraser.)

FIG. 15. (Case III.)

High-explosive shell injury of the ear. Horizontal section, No. 203, × 38 diam. (iron-hæmatoxylin). Shows condition of Corti's organ in apical and middle coils; except for the absence of the hair cells, due to faulty fixation of the specimen (?), there appears to be little change in the acoustic papilla.

Microscopic Examination.

Drumhead.—This is ruptured in the lower part posteriorly, but the edges of the perforation are for the most part glued together by blood clot (fig. 25).

Tympanic Cavity.—There is hæmorrhage in the marrow spaces, in the roof of the tympanic cavity, and there is a little blood in the tympanum itself

in the angle between the drumhead and the malleus. The ossicles and the joints are normal, but there is slight hæmorrhage in the canal for the tensor tympani. On the inner wall of the tympanic cavity there is some effused blood around the geniculate ganglion. The structures in the oval and round windows are normal.

Labyrinth Capsule.—There is a small hæmorrhage in the fossa subarcuata, but otherwise the labyrinth capsule is healthy.

FIG. 16. (Case III.)

High-explosive shell injury of the ear. Horizontal section, No. 203, × 38 diam. (iron-hæmatoxylin). Shows normal (?) condition of Corti's organ in upper part of basal coil.

Labyrinth Contents: (1) *The Cochlea.*—The outline of Corti's organ is preserved, but many of the cells—not only the hair cells but also the supporting cells—have disappeared, so that Corti's organ appears rather like a ghost or skeleton. The epithelial cells of the stria vascularis appear to be detached from the spiral ligament in the basal and middle coils. There is blood in the scala tympani in the region of the round window,

(2) *Vestibule.*—The otolith membrane of the saccule is separated and the neuro-epithelium irregular. The aqueduct of the vestibule is normal.

(3) *Canals.*—The cupula of the superior canal is separated, but the ampulla of the posterior canal is normal.

Internal Meatus.—There is extensive hæmorrhage in the fundus of the internal meatus, and there is also some blood in the bony canal which contains

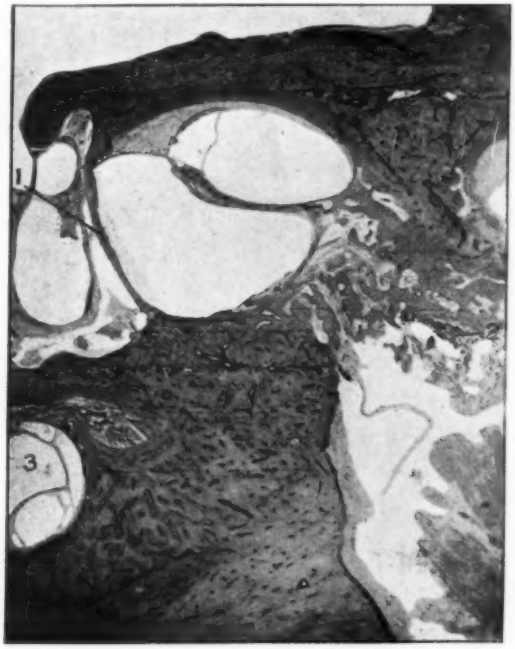

FIG. 17. (Case III.)

High-explosive shell injury of ear. Horizontal section, No. 265, × 11 diam. (hæmatoxylin and eosin). Shows region of round window and fundus of internal meatus with hæmorrhage. 1, Membrane of round window; 2, hæmorrhage in internal meatus; 3, ampullary end of posterior vertical canal.

the branch of the vestibular nerve for the crista of the lateral canal. There is also a considerable amount of blood pigment in the internal meatus.

(*See* figs. 18 to 24.)

Summary.

The changes in this case of ear injury due to grenade explosion are fairly definite and include a recent rupture of the tympanic membrane, the edges of which are partially glued together by fibrin. Further, there are hæmorrhages in the tympanic cavity, in the marrow spaces in the roof of this cavity, around the geniculate ganglion and in the fossa subarcuata. There is also bleeding in the scala tympani of the cochlea in the region of the round window. There is a

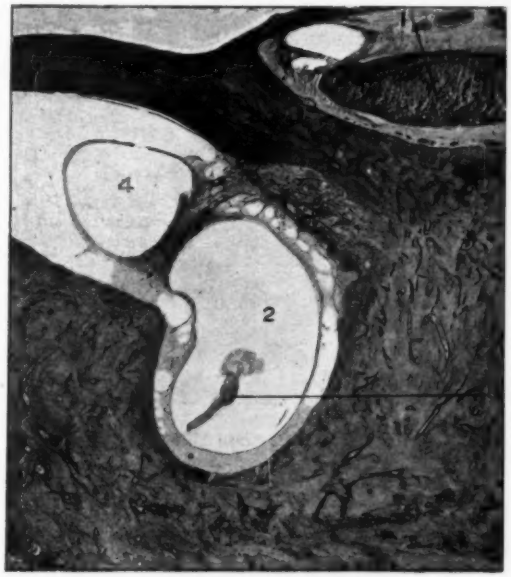

FIG. 18. (Case IV.)

Injury of left ear due to bursting of rifle grenade. Horizontal section, No. 125, × 14 diam. (hæmatoxylin and eosin). Shows crista of lateral canal and displaced otolith membrane of utricle. 1, facial nerve; 2, upper part of utricle with, 3, detached otolith membrane; 4, ampullary end of lateral canal.

marked hæmorrhage in the fundus of the internal meatus. The otolith membrane of the saccule is separated and the neuro-epithelium is irregular, while the cupula of the superior canal is also separated. Although the outline of Corti's organ is preserved, the hair cells and some of the supporting cells cannot be made out, so that the organ appears ghost-like. (These appearances in the neuro-epithelium of the labyrinth may possibly be due to post-mortem change,

FIG. 19. (Case IV.)

Injury of left ear due to rifle grenade. Horizontal section, No. 225, × 14 diam. (hæmatoxylin and eosin). Shows oval window with stapes; displacement of otolith membrane of saccule; desquamation of neuro-epithelium. 1, foot-plate of stapes; 2, neuro-epithelium of saccule; 3, detached otolith membrane.

FIG. 20. (Case IV.)

Rifle grenade injury of left ear. Horizontal section, No. 230, × 48 diam. (iron-hæmatoxylin). Shows, 1, shrunken (?) condition of cells of spiral ganglion of middle coils.

FIG. 21. (Case IV.)

Rifle grenade injury of left ear. Horizontal section, No. 230, × 48 diam. (iron-hæmatoxylin). Shows irregular condition of neuro-epithelium of saccule and loss of otolith membrane.

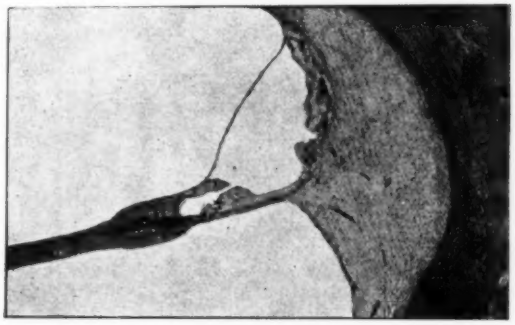

FIG. 22. (Case IV.)

Injury to left ear due to rifle grenade. Horizontal section, No. 230, × 48 diam. (iron-hæmatoxylin). Shows condition of Corti's organ in upper part of basal coil. The epithelial cells are irregular and do not stain well, and the stria vascularis is desquamated.

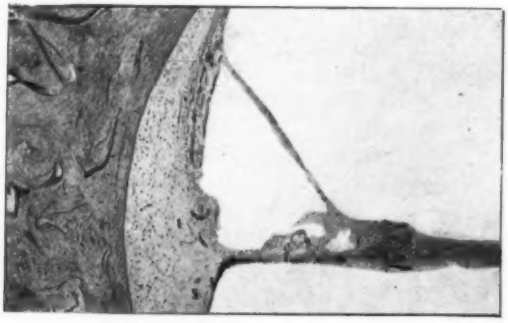

FIG. 23. (Case IV.)

Injury to left ear due to rifle grenade. Horizontal section, No. 230, × 48 diam. (iron-hæmatoxylin). Shows condition of Corti's organ in lower part of middle coil. The epithelial cells and the vessels in the bony spiral lamina stain badly.

FIG. 24. (Case IV.)

Cochlear canal with Corti's organ from middle coil of cochlea in an old man aged 80, who suffered from nerve deafness. It will be seen that the acoustic papilla is well formed. (This specimen is shown for comparison with those obtained from Cases III, IV, V, and VI, in which the ears had been subjected to concussion caused by shell or rifle-grenade explosion.)

but in our opinion, they are more probably due to "degenerative neuritis" of the nerve structures of the inner ear.) In this case the stress of the explosion appears to have been exerted on the posterior inferior part of the drumhead, the region of the round window and the fundus of the internal auditory meatus.

FIG. 25. (Case IV.)

Injury to left ear due to explosion of rifle grenade. Horizontal section, No. 305, × 9 diam. (hæmatoxylin and eosin). Shows, 1, rupture of drumhead with blood clot on anterior margin; 2, scala tympani just internal to round window membrane with thin layer of hæmorrhage on the walls; 3, fundus of internal meatus with hæmorrhage.

CASE V.—SHELL EXPLOSION.

The patient suffered from severe concussion and the right side of the head and body presented multiple wounds.

Naked-eye examination of the ear showed a plug of wax in the right external auditory meatus. The right drumhead appeared lustreless, opaque and indrawn after the wax had been removed.

Microscopic Examination.

The drumhead appears to be unusually thick, but is otherwise normal.

Middle-ear Spaces.—There is slight hæmorrhage in the tubal part of the tympanic cavity and on the inner wall of the middle ear. The tympanic

ossicles and joints are normal. The footplate of the stapes shows a congenital dehiscence in the bone, filled with connective tissue. The niches of the oval and round window are healthy.

Hollow Spaces of the Labyrinth: (1) *Cochlea.*—There are small round masses of hyaline substance in the scala tympani and scala vestibuli, but these are probably of little importance. Corti's organ shows a normal outline in all coils. The hair cells cannot be made out, but this of course is not unusual in

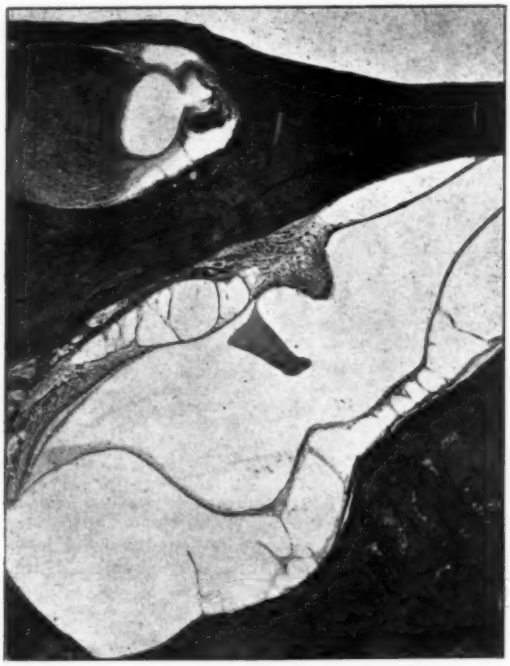

FIG. 26. (Case V.)

Injury (?) of right ear due to shell explosion (the right meatus contained a large plug of wax). Horizontal section, No. 95, × 19 diam. (hæmatoxylin and eosin). Shows displacement of cupula of lateral canal—artefact?

post-mortem specimens. The cochlear opening of the perilymphatic aqueduct is normal, but towards the cranial end the aqueduct contains a little hæmorrhage.

(2) *Vestibule.*—The otolith membrane of the utricle is raised up from the neuro-epithelial cells (artefact?). The saccule and its otolith membrane are normal. The ductus endolymphaticus is healthy.

(3) *Canals.*—The cupula of the external canal is misplaced, as is also that of the superior canal; otherwise these canals are healthy. The crista of the posterior canal is healthy.

Internal Meatus.—The facial nerve is healthy. There is some hæmorrhage in the fundus of the meatus around the cochlear nerve.

FIG. 27. (Case V.)

Injury (?) of right ear due to shell explosion. (The right meatus contained a large plug of wax.) Horizontal section through the vestibule. Shows the normal condition of the saccule with otolith membrane intact.

Summary.

The changes in this case are very slight. This may possibly be explained by the presence of a plug of wax in the external meatus. There are small hæmorrhages in the tubal part of the tympanic cavity, in the canal for the tensor tympani, in the cranial ends of the fossa subarcuata and perilymphatic aqueduct, and in the fundus of the internal meatus. The neuro-epithelium of the labyrinth shows little change, but the detachment of the otolith membrane

of the utricle and of the cupulæ of the lateral and superior canals is worth recording. If a similar condition were found in the labyrinths of other patients who had suffered from shell explosion, these conditions might be regarded as of importance.

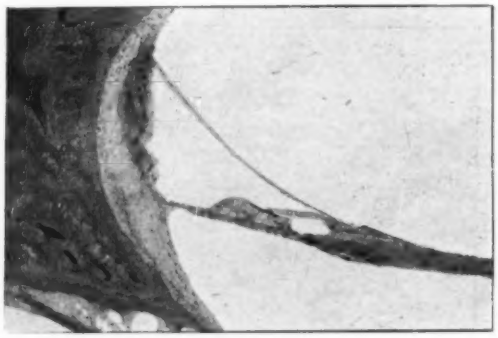

FIG. 28. (Case V.)

Injury (?) to right ear due to shell explosion. (The right external meatus contained a large plug of wax.) Horizontal section through cochlea showing normal (?) condition of Corti's organ in middle coil.

CASE VI.—INJURY BY HIGH-EXPLOSIVE SHELL.

This soldier caught the full blast of a high-explosive shell which burst close to his left side and shattered this side from head to foot.

Naked-eye Examination.—Left tympanic membrane shattered, only the outer margin remaining attached to the annulus. The malleus appears to be pushed inwards with a piece of membrane attached to it. The long process of the incus can be seen through the perforation. The inner wall of the tympanum presents a reddish-brown appearance, but there is little evidence of blood in the antrum. The superior canal appears normal when opened to admit the fixing fluid.

Microscopic Examination.

Tympanic Membrane.—There is a large gap in the drumhead in front of, below and behind the handle of the malleus, but there is no rupture of Shrapnell's membrane. The lower part of the handle of the malleus, with a piece of membrane attached, is in contact with the promontory.

Middle-ear Spaces.—The *Eustachian Tube* contains hæmorrhage. The head of the malleus is healthy and the joint between the malleus and incus is normal as is also that between the incus and stapes. The stapes footplate appears to be somewhat displaced and the posterior part of the annular

ligament is ruptured (artefact?). The posterior part of the footplate of the stapes is tilted outwards towards the tympanic cavity (artefact?).

Hæmorrhage is present on the inner wall of the attic and also between the short process of the incus and the outer wall of the aditus. Free blood is also seen in Prussac's space. In the lower part of the cavity the inner wall shows a layer of hæmorrhage and the niches of the oval and round windows are full of blood. The lining membrane of the tympanic cavity is congested and slightly swollen. The sinus tympani also contains blood. The membrane of the round window appears normal.

FIG. 29. (Case VI.)

Injury of left ear due to high explosive shell. Horizontal section, No. 285, × 15 diam. (hæmatoxylin and eosin). Shows rupture of drumhead in front of and behind the malleus. The niche of the oval window and the hollow of the stapes contain blood. 1, posterior part of ruptured drumhead; 2, malleus; 3, incus; 4, blood in hollow of stapes; 5, facial canal (the nerve is displaced—artefact); 6, chorda tympani.

Hæmorrhage is present in the cells in the roof of the antrum and in the border cells between the antrum and external meatus. There is a little free blood in the floor of the aditus.

Labyrinth Capsule.—There is some hæmorrhage in the canal for the great superficial petrosal nerve. The vessels of the fossa subarcuata are dilated.

Labyrinth Contents: (1) *Cochlea.*—Owing to a fault in the first embedding of the specimen, air entered the hollow spaces of the labyrinth, and in the second embedding the evacuation apparatus was used to extract the air. It is therefore impossible to speak with any degree of certainty regarding the condition of the nerve structures. The intravestibular and apical portions of the cochlear canal are more normal than the middle portions, but this may be due to the fact that the latter was more affected by the suction apparatus,

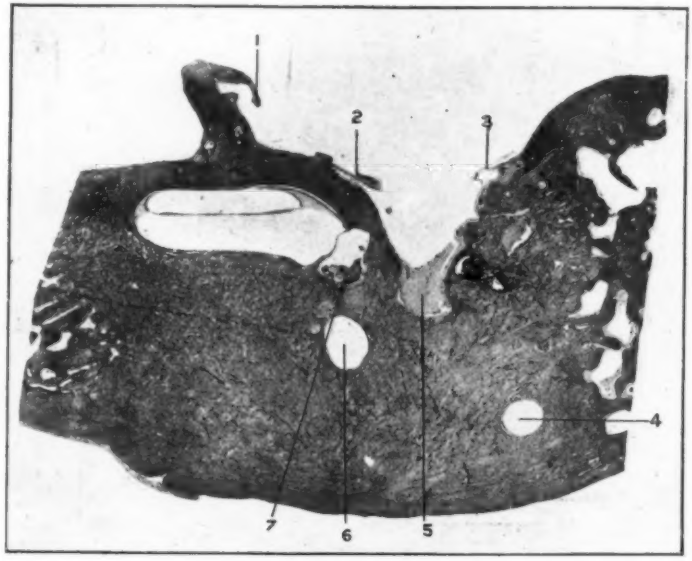

FIG. 30. (Case VI.)

Injury of left ear due to high-explosive shell. Horizontal section, No. 410, × 6 diam. (hæmatoxylin and eosin). Shows large rupture of drumhead. The central part of the membrane is in contact with the promontory. There is hæmorrhage in the niche of the round window. 1, anterior part of ruptured drumhead; 2, central portion of drumhead in contact with promontory; 3, posterior part of drumhead; 4, smooth end of posterior vertical canal; 5, blood in sinus tympani; 6, ampullary end of posterior canal; 7, blood in niche of round window.

which was only applied after the basal coil of the cochlea had been opened. The cochlear opening of the perilymphatic aqueduct is normal.

(2) *Vestibule.*—The neuro-epithelium of the utricle and saccule appears healthy. The aqueduct of the vestibule is normal and the ductus endolymphaticus is quite healthy.

(3) *Canals.*—The superior canal is normal except that in the ampulla the epithelium is irregular (artefact). The cristæ of the external and posterior canals are normal.

The internal meatus and nerves show no hæmorrhage nor other abnormality.

Summary.

In this case the force of the explosion seems to have been expended in rupturing the drumhead, so that the structures of the inner ear, along with the nerves in the internal meatus, have escaped. Owing to a fault in the first embedding of the specimen, air was present in the hollow spaces of the labyrinth, and it is therefore unwise to be dogmatic as to the condition of the membranous labyrinth. The rupture of the saccule and of portions of the membranous cochlea are probably artefacts produced by the use of the suction apparatus. The rupture of the posterior part of the annular ligament may also be due to the same cause.

Conclusion.

The only changes of importance found in the four cases of "explosion" injury of the ear are: (1) Rupture of the drumhead (Cases III, IV, and VI) and hæmorrhage into the middle ear spaces; (2) hæmorrhage in the fundus of the internal meatus in three of the four cases. In Cases III and V the neuro-epithelial structures of the labyrinth appear to be normal. In Case IV the changes are possibly of "post-mortem" origin, but they appear to us to be due rather to an early stage of "degenerative neuritis," while in Case VI they are caused by a fault in the preparation of the specimen. It seems quite possible that in many cases of "shell" or "explosion" deafness we have to deal with a functional affection as suggested by Milligan and Westmacott. On the other hand, rupture of the drumhead and hæmorrhage into the middle ear spaces must cause a certain loss of hearing; while hæmorrhage in the fundus of the internal meatus may give rise to deafness, tinnitus, giddiness, and other symptoms of an inner ear lesion. It may be that the "blow" to the ear due to shell explosion, and the associated loud sound, paralyses the delicate nerve endings of the auditory apparatus, but we cannot claim to have demonstrated this microscopically, except possibly in Case IV. As we have already said, microscopic examination of the ears from cases of shell deafness, which die from other causes years after the injury, would be of great value in clearing up the pathology of the condition, and especially in throwing light on the question of "degenerative neuritis" of the cochlear apparatus.

The writers wish to thank Mr. Richard Muir for the photomicrographs which illustrate this paper. They also acknowledge a grant from the Carnegie Trust in payment of the expenses incurred in reproducing the illustrations.

Colonel BIRKETT, C.B.

I cannot offer any suggestions on the discussion which has been opened by yourself, but I think that perhaps Dr. Gordon Wilson, who has been making a special study of this work, may have a great deal more to say on the subject than I can. The number of ear cases in our hospital, which contains 2,000 beds, is very great: I have a ward of seventy-five beds set apart specially for eye, ear, nose and throat cases. The class of ear cases which presents itself there is very largely the chronic suppurative variety. We do see a large number of cases of rupture of the tympanic membrane following high explosives. As to the situation of these ruptures, it has not seemed to me that there is any particular predominance of any one site. What we have noticed, and what we have tried to impress on the authorities there, is that interference with these ears, in the first place, should not be carried out at the Front. The patients nearly always come down with a history of having had the ears syringed. Last July, when we had these cases early, they came to us without having had this interference. We could then take care of them from the first, and we treated them as you suggested, Sir, and the result has been a satisfactory healing in the majority of cases. Where the rupture has been of considerable duration I found that nitrate of silver, 25 gr. to the ounce, applied to the edges of the rupture, stimulates the healing of it, so that in from six to eight weeks there has been complete closure, with marked restoration of hearing.

As to the psychical cases, they are, perhaps, the most pathetic we have to deal with. These men are broken in spirit, and their nerves are entirely shattered, and this condition is nearly always associated with mutism. It is, therefore, impossible to get any accurate results from our tests. We begin by encouraging these men to hear and speak. Often, however, those methods do not succeed, and then we try to shame the men out of their disability before their companions-in-arms. Sometimes those means are successful. One method was letting off an alarm apparatus suddenly: this shook one man so much that he eventually heard. I have nothing new to say about the subject, or anything to add to the pathology.

Dr. Gordon Wilson.

It has been my good fortune, during the last six months, to be associated with the Canadian Hospital at Folkestone, and I have had many cases of deafness associated with labyrinthine troubles; I have records of over 100 cases of nerve deafness. Recently I was sent to France to see recent cases of concussion-deafness there. I have not yet had time to examine my specimens, but I hope to do so when I get back.

We divided our cases into three groups: (1) nerve deafness; (2) cases of men with what we call a fixed idea that they cannot hear; (3) malingerers. Rupture of the drum may take place at any part of it; I have seen several cases involving the membrana flaccida: in one case it was driven in against the promontory, with complete rupture of the drum. In France it was curious to note how many of the men had no hæmorrhage from the ear, because, it was said, the injury had been so slight. While I was at the Ypres salient and on the Somme I probably saw 250 cases of shell shock. Only fifty of those complained of deafness, and of those fifty only seventeen had actual nerve deafness, a very small proportion. But at that time things were quiet on the Somme—at the end of December and in the month of January.

With regard to the cases with a fixed idea, rapid recovery occurred under hypnotism. Some of the cases recover if cold water is run into the ear. We never use the electric current, because if we do the result is an irritation of the vestibular nerve, and the production of intense vertigo.

With regard to the treatment of our cases, we ascertain whether the labyrinth has any activity in those in which there is complete deafness on one side or both. In all the cases we had at Westcliffe there was none in which mutism was not present, and all of them had been under otologists for periods ranging from two to twelve months, and had been sent to us at the end, particularly Canadians. We first run cold water into the ear, and get a caloric reaction. If we do not get that we do not trouble. If we do we proceed, always using tuning forks. If we use the C, or the C_1 fork against the ear, we may not get a reaction, but by using both, and thus having a summation of stimuli, we get it. In totally deaf cases we proceed to use resonators. We get tubes like speaking-tubes in the ear. All this will be published in full later.

One thing which has interested us very much I would like to mention.

We have had cases of wounds of the cerebellum, and the question has arisen in connexion with the differential diagnosis of labyrinthine disease from cerebellar tumour. It has been our good fortune to keep some of our cases under observation for several months, and we have been able to show that pure cerebellar injury results in a form of nystagmus which is different from that found in association with labyrinth cases. I need not proceed to tell you how that is the case. I was very pleased to see Colonel Gordon Holmes when I was in France, and to learn that he had a large number of ward cases which bore on that fact. There is nothing more important in connexion with the ear than to be asked the question: Is that a cerebellar case or is it the beginning of a labyrinthine case? I think the war records will show that some of our most difficult cases of cure have been those in which a missile has cut the facial nerve outside the ear, or in which there was complete deafness which we could not touch at all. When I was in Paris I was much interested in noticing that the same thing may be said of the war experience in France. I wonder whether anyone has noticed, in the deaf cases following trauma, a peripheral limitation of the field of vision as well as a peripheral limitation of hearing.

Mr. C. E. West.

I have found exactly the same thing as the last speaker just mentioned—namely, that the auditory field is contracted at both ends in these cases, and more or less symmetrically. I do not know whether in many cases the speaker found contracted visual fields as well. [Dr. Wilson: Yes.] The analogy of the two struck me so much that I use it as a metaphor in teaching.

I have seen comparatively few cases of direct injury of the ear while doing my war work, and that has surprised me. There have been a certain number of injuries of the pinna, cicatricial contraction, and so on, but they have been of no particular importance. Generally, the collateral injuries are so much more important that one has to relegate the question of the ear to a secondary place.

One group of cases has struck me particularly—namely, glancing wounds of the temporal region about the ear without fracture, apparently a scalp wound only, but with the pericranium involved. In each one of those cases there was gross labyrinthine deafness. I saw, this afternoon, a gentleman who, years ago, had been shot at by a Dacoit in India, and received an exactly similar wound, and had just the same

deafness on one side—in each case the same side as the injury, the other ear not being affected at all. How far it is permanent we do not know, because we have not the opportunity of following out indefinitely the cases which occur in the War, but the deafness is prolonged.

And we come across a large number of people who are said to be suffering from explosion deafness and who, manifestly, are suffering from continued old chronic middle-ear suppuration without labyrinthine deafness or symptoms, or who have discovered, as the result of their own overhauling, after being blown up, that they have middle-ear deafness and are attributing it to shell shock. A large proportion of the cases I have seen have been of that sort.

Another interesting thing is that there is a fashion in shell-shock effects. In the earlier days of the War there were a large number of cases of pseudo-labyrinthine deafness — people who recovered spontaneously or under some form of suggestion. Recently, at the First London General Hospital, these have been practically absent, and in their place there has been a stream of aphonics. I think these are largely due to suggestion: if a man thinks he will be rendered voiceless by being blown up, he is found to be so if he encounters that accident. With regard to people who, apparently, have true labyrinthine deafness, I do not know whether other surgeons succeed better, but I am hopelessly in the dark in regard to prognosis in these cases. A large number appear to recover almost completely, and the majority seem to get back useful hearing, slowly and gradually. A certain proportion of them seem to make no recovery. If anyone can tell me any bases for prognosis I shall be very grateful.

Dr. Dundas Grant.

Like Mr. West, I have been surprised that we do not see more cases of clear-cut labyrinthine injury. A large number of them consist of very ordinary cases of chronic suppuration or chronic catarrh, down to mere impacted cerumen. But there are many about which certain generalities may be stated.

My experience has been the same as that of our President and others: that where the membrane has been ruptured the involvement of the labyrinth, as a rule, has been very slight, in some cases the labyrinth has even escaped, though one of Mr. Fraser's sections seems to disprove that. That case appears to be an exceptional one.

I should like to know whether many members have seen what I

should call an " in-driven " membrane. I have seen a few in which the membrane has been driven in and so closely related to the ossicles that the appearance was as if the ossicles were sculptured out, just as in an extreme case of relaxed membrane. In these cases the margin of the tympanum has been well defined.

With regard to the question whether a perforation with suppuration is recent or of old standing, or whether it is a recent traumatic rupture with subsequent suppuration, I think the best guide is the condition of the Eustachian tube. If it is quite patent, it is reasonable to think a traumatic injury of the drum is a recent one. If the suppuration has continued a long time and the Eustachian tubes are not very patent, it is impossible to say from this whether it is a chronic case or a traumatic rupture.

With regard to prognosis, of course when the caloric test is negative, the prognosis is probably at its worst. Another point is the upper range of audition, for when that is considerably lowered, the prognosis, as a rule, is bad. When that upper range is fairly well preserved, the prospects are good. I have gathered some statistics from thirty-one cases in which it has been tested. It was normal in twenty-five of them, there was slight lowering in three, and very considerable lowering in the remainder. In those in which it was preserved, the improvement was marked or very considerable. But in one case, at all events, in spite of that upper limit being preserved, there was no improvement, and it was found that there was a slow reaction to the caloric test. There were four cases in which the upper limit was preserved and there was no improvement.

With regard to the cases of deafness, a number of them came with the complaint of " deafness," some of them had nerve deafness, and the question arose as to whether there was nasal obstruction. I thought I should find it in a large number. Of fifty cases there was marked deflection of the septum in eleven, but thirty-one had nothing of the sort. Four had adenoids. Not all the cases of deflected septum caused any considerable obstruction.

With regard to the caloric tests, I would remind you of the apparatus for producing cold in the ear which I have before exhibited to the Section, and which has apparently been taken up by the Army otologists in France as a regular official test. It is a tube of copper, covered with a little cotton moistened with ethyl chloride. A current of air is driven through it, and as soon as it is at its coldest, as tested on one's own cheek, the patient's head is thrown back at an angle of 60° and

he is told to turn his eyes to the opposite side, when nystagmus should be produced in from twenty-eight to thirty seconds. This obviates the dangerous practice of injecting cold water into the ear, in which a perforation may already exist. It has also been taken up by Moure in Bordeaux. The point through which the air enters the tube should be very small, otherwise the current is jerky. Dr. Paterson, of Cardiff, said it would be a good thing if we had an equally portable apparatus for making the hot test. I show you an apparatus which will do this very quickly. Mr. Clayton Fox has helped me in testing many cases, and we find we get nystagmus to the same side in about fifteen seconds. Possibly some members may devise an improvement upon it. Perhaps the bottle might contain a saturated solution of sulphate of soda, which is heated, and allowed to solidify again, and heat thus generated. In cases in which you wish to get nystagmus to the same side rather than to the opposite side—there being one in that direction already—this solves what has hitherto been a problem.

With regard to the cases of psychical deafness, much can be accomplished by re-education. You get them to hear a simple vowel, but you must not worry them too much, or you will throw them back into their nervous state. I do not think Colonel Birkett drew a line of demarcation between psychical deafness and simulated deafness. No doubt the reason he has not done so is that there is no hard-and-fast line: there are so many cases which blend both. I had the case of an officer who said he was quite deaf. We tried the noise machine, and he rose to it at once. That might be called simulated, but I think it is psychic, because he had not been a neuropathic, but had been so shaken up that he was reduced to the extremity of exaggerating his complaint, which he had only in a mild form, and which probably existed when he was "buried." It passed off. I think the majority of the psychical cases have no simulation about them. A test which helps us is the pupil reaction. We know that any sensory stimulus will cause dilatation of the pupil; for instance, while the pupil is carefully observed, we blow a whistle, or, in an extreme case, a motor-car hooter behind the patient, and watch for dilatation of the pupil. But though a man may rise to that, it does not necessarily follow that he is shamming. To test it with success, we must have the pupil contracted by light, and see whether it dilates on the sound being produced.

I should like to put two questions, which I do not feel equal to anwering myself: (1) What is the degree of deafness which justifies us in looking upon a man as being ineligible for general service? We see

how one man who cannot hear the watch makes progress, while another may be very stupid. Is there any standard? I am thinking of listening-posts. The second question is, what are we to do with those who have suppuration of the middle ear? Must we send them to the Front or not? My friend Mr. Kisch, who was in Boulogne for a long time, said that no man who had even had a radical mastoid operation should be sent to the Front. But I think that is carrying it too far. I showed a case, about sixteen years ago, in which I had preserved a cholesteatomatous membrane. The patient disappeared until last year, when he came back as a stalwart sergeant, who had been all through the War, and it was only discovered that he was deaf in one ear when he got injured in the other. Another case was that of a boy whom I showed here, who had suppuration of the middle ear. I opened a cerebellar abscess and the bulb of the jugular vein. He has been eighteen months in the War as a driver of the R.F.A.

I have spoken at this length about these things because there are some cases about which I feel a difficulty, and I shall be glad if other members can help me.

[Major SHUTER: Was the upper limit of audition taken by Dr. Grant by air conduction, or by bone conduction? And with what instrument?]

Dr. DUNDAS GRANT: I take it with as good a specimen of Galton's whistle as I can get—the original whistle. It does not go as high as the monochord. I exclude as far as possible the function of the opposite ear. It indicates the hearing for a closed pipe of so many millimetres in length, and if the hearing is 1·5, 1·8 or 2, I pass the case as one of good preservation. I think that whistle is sufficient for all clinical purposes.

Dr. C. E. JONES-PHILLIPSON.[1]

I hope I may be permitted a few remarks as one who has looked after the ear cases for the Third Army, having been doing that work for nine months. I had the good fortune to be located at Stationary Hospital No. 12, where the Surgeon-General concentrated ear work, allocated forty-four beds (indoor), and gave out-patient accommodation for eighty. All the work of the Third Army was passed through this Department, and I made notes of 1,172 cases examined during that

[1] Late Medical Officer in Charge, Ear, Throat, and Nose Department, Third Army No. 12 Stationary Hospital, British Expeditionary Force.

period. As will be seen from the abstract I analysed 100 cases of war injury of the ear, in which there were thirty-one cases of lacerations. I saw all my cases within twenty-four to thirty-six hours of their coming out of the trenches, some on the day of receiving the wound. A previous speaker, and a part of the President's address, laid no particular stress on the site of the laceration, but from my cases I regard that as very significant. In thirty-one cases the laceration occurred in the anterior-inferior quadrant in twenty; in the postero-inferior quadrant in six; in the inferior quadrants—i.e., between the two—in one; in the umbo in two; in the posterior quadrants across the two in one; in the antero-superior quadrant in one. It has also been asked whether any note had been made of rupture of the membrana flaccida. I had such a case, which is noted under the head "blood in the middle ear." No. 31 had a ruptured membrana tympani in the antero-superior quadrant, high up. There was a slow oozing of sero-sanguineous discharge for several days. Patient went to the base shortly after, and I thought it possible he had a fracture through the tegmen. He also had pains in the back of his neck and over the mastoid, and his face had been well peppered with fragments. I had four other cases of distinct blood-clot in the middle ear. In the case mentioned it was with rupture of the membrana tympani, in another without. The patient volunteered the information that he could taste blood in his mouth, and I thought it came through the Eustachian tube; I could not see it by posterior rhinoscopy.

Many also volunteered the information that they were knocked out or were unconscious, some remaining so for one or two hours. I had this verified in many cases. After this state had passed off the men were in a dazed condition, and often could not walk unassisted. The more profound cases had lost the power of speech, and two or three of the cases could not see. The major conditions often passed off fairly quickly, leaving dizziness and giddiness, which lasted for a long time. These cases I examined with great care. I found they showed a horizontal or rotatory nystagmus on turning the eyes to either side; I do not mean on turning the eyes to the extreme degree permitted by the orbit, because it will be produced in anyone by that means. In this connexion I instance cases No. 23, 47, 48, 52, 56, 78, 83. It should be noted which side was most affected by the explosion. When it occurred on the parapet in front of the patient, he has a double, right and left, spontaneous horizontal nystagmus, or right or left according to the side affected.

One more little matter I would like to bring before you has reference to caloric tests. They require very much more water to prove a functionating or non-functionating labyrinth in the cases at the Front than in civil life, because these ears have been profoundly disturbed. You cannot tell whether a labyrinth is functionating unless you continue your test with hot or cold for three times as long as normal. In only two of my cases out of 100 did I find the caloric test brought no response—namely, Nos. 85 and 86. In these two cases I used as much as two and three quarts of water; they had intact membranæ tympani. Those were the cases which were most profoundly affected, both in regard to tuning-fork reactions and in response to the caloric test. The nystagmus varied—it was definitely "there" in all except Nos. 85 and 86—often very quick and feeble or very slow and feeble. In one case the nystagmus was very coarse, only comparable with the movements of the pendulum of a big clock—slow, with long excursions. In a few the nystagmus was quickly induced and very violent. The patient looked deadly pale, with extreme giddiness, faintness, and vomiting; in two the effect was so prolonged that the cases were unable to return to the ward for fifteen to twenty minutes.

With regard to movements produced by caloric tests, I do not wish to be understood to say that every case requires to be tested with a large amount of water to prove function of the labyrinth, but some labyrinths are profoundly depressed by the concussion, and are only slowly responsive. There are cases in which there is extreme activity, as in that of the lieutenant I have mentioned.

An Analysis of 100 Cases of War Injuries of the Ear.

The War has afforded the opportunity to further the knowledge of injuries to the internal ear. Many obstacles are in the way, and much valuable information will not be recorded.

(1) Many cases were not examined owing to the severity of other injuries; these were principally head cases under the care of surgical specialists, and one was reluctant to propose an examination and do the necessary tests, which might have produced disturbance and perhaps have retarded the progress of the case.

(2) Post-mortem evidence of damage to the internal ear might have been found, but these evidences would have been unaccompanied by definite symptoms complained of by the patient, as these cases were nearly always comatose.

(3) The transfer from firing line to base, and base to England, often quickly and always unexpectedly carried out, prevented me from repeating tests at intervals, and I was seldom able to watch a case for longer than fourteen days.

(4) The difficulty in tracing these cases later and in obtaining anything of value in the replies when obtained.

(5) In a hurriedly recruited army large numbers of men start military life as practically deaf men, and the increased loss said to be due to shell concussion can never properly be gauged.

External Ear.

Various degrees of laceration of the external ear are commonly seen—slits, perforations, or nearly complete severance of the auricle. They healed well and quickly. Two cases of severe perichondritis of the auricle with abscess formation were seen. Three cases of external ear injuries are here recorded showing no middle or internal ear damage.

(1) Private R. R. Cleaning up a billet by sweeping rubbish into a fire, a cartridge was swept in and exploded. A portion of the "fang" of the cartridge case went into the external auditory canal and was impacted there. There was no injury to the membrana tympani—merely an abrased canal.

(2) Private H. R. Bullet entered at the posterior border of left mastoid process, say ½ in. from tip, above and behind it; opening seen in pharynx about 1 in. to mesial side of posterior pillar of left fauces, passed obliquely across the nasopharynx, striking posterior end of right middle turbinate and out of right eye. Right antrum was full of blood and right eye destroyed.

(3) Private P. W. P. An aerial torpedo burst and wounded him deeply at the back of the neck, behind right ear and lower jaw. Wound extended from the base of right mastoid process to centre of right sternomastoid—posterior five-sixths of origin of right sternomastoid destroyed—anterior one-sixth remains. Recurrent paralysis of right cord, complete paralysis of right half palate; affection of lower half of facial, and of cervical and brachial plexus; X-ray showed fragments of shell deep down against cervical vertebra.

Notes of over 100 ear cases were made of patients who definitely attributed their deafness or increased deafness to the effect of "guns," "explosions," "bombardment," "being buried." An examination of the ears, nose, and nasopharynx was made in each case. Seventy per cent. of the cases examined showed pre-existing ear, throat and nose conditions: defects due to otitis media suppurativa, 34; defects due to C.D.C., 22; defects due to nose and nasopharynx, 17.

In no case was the nasopharyngeal airway considered faulty or worth

noting unless it was a distinct abnormal condition as shown by nasal obstruction, due either to deflected septum plus turbinate hypertrophy —hypertrophy of the turbinates alone—adenoids alone or in conjunction with one or other of the former, and could be considered as a predisposing cause to a diseased condition of the ear.

It is my intention to compare the effects of explosion on the membrana tympani, middle ear, and internal ear: (1) In normal conditions; (2) in diseased and predisposing conditions.

Lacerations of the membrana tympani were seen in thirty-one cases: in normal conditions, fifteen; in diseased and predisposing conditions, sixteen.

Lacerations in pre-existing conditions are accounted for as follows: Conditions of nasal obstruction, three; conditions of otitis media suppurativa, healed by scar, five; conditions of C.D.C., four; conditions of nasal obstruction and adenoids, one; conditions of nasal obstruction and otitis media suppurativa, one; conditions of otitis media suppurativa, with loss, one; conditions of adenoids, one.

Site of laceration in total number of thirty-one cases: anterior inferior quadrant, twenty; posterior inferior quadrant, six; inferior quadrants, one; umbo, two; posterior quadrants, one; anterior superior quadrant, one.

Laceration of membrana tympani: (*a*) intact membranes, twenty-four; (*b*) with loss or scar, seven.

Hæmorrhages into membrana tympani (intra-membranous): (*a*) intact membranes, sixteen; (*b*) with loss or scar, six.

Lacerations were irregular, tears or flaps. Their edges were always swollen and red. They did not heal well in the large majority of cases. The edges or much larger portions sloughed, especially when the laceration was flap-like. Many cases were seen within twenty-four to thirty-six hours of the damage. The parts were gently syringed with lot. bor.—or lot. hydrogen peroxide — 10 vol. solution diluted four times with hot water, and a hot alkaline nasal wash prescribed. The surrounding ear parts were painted with tinct. iodi and 3 or 12 per cent. glyc. acid carbolic drops into ear, calomel, mist. alba and the patients were kept in bed.

A subacute or acute otitis media, followed by suppuration, was present in the majority. When we know that very few wounds received in this war heal by first intention, such healing cannot be expected in ear cases, which are extremely dirty in the majority of cases. Intra-membranous hæmorrhages frequently sloughed and led to similar conditions.

Blood in the Middle Ear (*vide* Nos. 39, 56, 60 & 76).

Four cases of firm blood-clot in the middle ear were seen, bulging the membrane outwards. In one instance the membrane was quite intact, the clot could be distinctly felt, and the membrane bulged over it as three air bulbs. In two of these cases the patient reported that blood came into his mouth, and that he could taste it, but it was not observed by posterior rhinoscopy.

No. 39.—Private T. L. Deafness since childhood; running ears; had acute pain in left ear. When on the battlefield he was in charge of the water-cart, and slept only 10 yards from the big guns. He complained of increased deafness and pain. There was blood-clot in the right posterior quadrants, and the left membrana tympani appeared as three air sacs covering a blue blood-clot; no perforation nor discharge, and it could be felt by a probe.

No. 56.—Private G. L. Had running ears when a child. Trench mortar shell burst near him and he was buried. Head became very giddy, with singing noises; he was like a drunken man; had staggering gait which passed off. On the right there were three intra-membranous blood patches; on the left a laceration, posterior inferior quadrant, and the middle ear was full of blood-clot. There was a very marked spontaneous horizontal nystagmus to left and also to right. His labyrinth right and left was hyper-excitable; his responses to the rotatory and caloric tests to right and left were easily produced and were normal but exaggerated. The tuning fork C_1 could not be heard by air conduction and was shortened negative right and left. Fork C_2 shortened negative right and left. Fork C_4 very short right and left.

No. 60.—Gunner S. S. A "Minnie" exploded 20 yards to the left of him causing deafness in right and left ears, and noises and discharge of blood from the left. On the right there was a flap laceration, anterior inferior quadrant of the membrana tympani; the left was bulged, bluish-red. There was a middle-ear blood-clot, and a laceration at the anterior inferior quadrant. The labyrinthine tests, rotatory and caloric, were both feeble. C_1, C_2, C_4, forks were all shortened, very short on the left.

No. 76.—Private H. B. Was cleaning the trench boards in a stooping position when a shell burst over him. There were shrapnel wounds of the face. He complained of left deafness; bleeding; giddiness; fainted at the time. On the left there was a laceration at the anterior inferior quadrant, an intramembranous hæmorrhage, and blood-clot in the middle ear. The rotatory tests produced a very brisk after nystagmus. The caloric tests right and left gave normal response, but hot irrigation of the left ear produced the tendency to fall to the right; hot irrigation of the right ear the tendency to fall backwards; patient became very pale and vomited violently. C_1, C_2, C_4 all heard short right and left, especially C_4. Weber to right.

In No. 31, with ruptured membrana tympani in anterior superior quadrant, high up, there was a slow oozing of sero-sanguineous discharge for several days (August 13, 1916, to August 21, 1916). He went to the base on August 22, 1916. I thought he possibly had a fracture through the tegmen tympani. He had, in addition, pains in the back of the neck and over the mastoid; his face had been well "peppered."

In No. 63 the left membrana tympani was bluish-red. Laceration at site of inferior quadrant; strands of tissue could be seen crossing the laceration; blood was exuding. The right membrana tympani had a calcareous patch at the same site. This may be an instance of a calcareous patch, the weak spot of a diseased membrane, having been driven in by the force of explosion.

The frequency of the complaint, "recurrence of discharge following shell concussion" in old conditions of otitis media suppurativa which have been dormant for years, seems more than a coincidence. The discharge in these recurrences, and following recent lacerations, was noticed in most instances on the third day.

Analysis of Symptoms.

In order of frequency:—

(1) Deafness, increased deafness, "dullness in ears."

(2) Noises in great variety (singing, buzzing, hissing, straining, thumping, bells, throbbing, ticking).

(3) Giddiness, dizziness, "dazed."

(4) Pain—soon passing off.

(5) Bleeding, at the time, or noticed soon after.

(6) Staggering gait, inability to walk, unconscious, dumbness, blindness.

Deafness was very marked soon after the explosion, always most on the side exposed to the full force; when the shell burst in front or behind a patient, both ears were affected. Patients who had been buried seem to have suffered more than others. The initial degree of deafness following the concussion soon passed off. The noises were often noticed only later, after the first degree of deafness had passed off. It was not often that increase in deafness was stated; as a rule, only the addition of noises to the previous condition. When the hearing showed further marked improvement, the persistence of the noises was a common complaint. Some patients reported being unconscious for from one to two hours; then feeling in a dazed condition, inability to walk unassisted, staggering gait; others that they lost the power to

speak, or could not see. All these maximum conditions appear to pass off quickly; the lesser conditions, giddiness and dizziness, were complained of for seven to ten to fourteen days—anyway while under observation. These cases showed a horizontal or rotatory nystagmus, or turning eyes to the right or left. It must be noted whether both right and left sides were likely to have been affected at the time, or right or left only. It will be noticed that when both sides are affected there was a nystagmus to the right and left, when one side was affected there was a nystagmus to the opposite side. (*Vide* Nos. 23, 47, 48, 52, 56, 78, and 83.)

Spontaneous Nystagmus.

In the following seven cases giddiness and dizziness were complained of:—

No. 23.—Rifleman F. J. S. This man has old losses of right and left membranæ tympani. He complained that gunfire caused extreme giddiness, followed by sweating and vomiting. He had a horizontal nystagmus on right and left sides. The rotatory and caloric tests showed a hyper-excitable condition of the right and left labyrinths. The forks were not badly heard. Rinne was negative right and left for C_2, and C_4 was normally heard right and left.

No. 47.—Private A. W. Deaf since a child; slept near the guns. The continual noise increased his deafness and caused buzzing noises and giddiness. He had a horizontal nystagmus right and left. Tuning forks C_1 and C_4 were shortened, C_2 practically normal.

No. 48.—Private M. Y. Had gunshot wound in right leg due to bomb explosion; deafness on left, straining noises, and giddiness. His membranæ tympani showed a condition of chronic dry catarrh. There was a rotatory nystagmus to the right. All the tuning forks were shortened on the left; Weber to right.

No. 52.—Lieutenant B. C. A. High-explosive shell burst a few yards from him, over him but more towards his left; he was stunned. Deaf right and left, noises in the head and pain for thirty-six hours. Both membranæ tympani were red. No sign of pre-existing disease. He has a spontaneous rotatory nystagmus to the left and horizontal nystagmus to the right. The rotatory and caloric tests showed a hypo-excitable condition of the labyrinth on right and left, the response was only a feeble one. The tuning forks for C_1 and C_4 were much shortened, C_2, although short, much less so in comparison. At the end of a week the right and left nystagmus had gone, C_1 and C_2 were now very slightly shortened; C_4 was still very short. The noises were much less, but present; giddiness had passed off.

No. 56.—Previously mentioned.

No. 78.—Lance-corporal J. B. A shell burst about 4 ft. from him, made him light-headed, and deaf in both ears. Was very giddy. Deafness to a large extent passed off. There was a laceration in posterior quadrant and discharge of pus, which had come on since the explosion five days ago. There was a horizontal nystagmus to the right and left. The tuning forks C_1, C_2, C_4, were all heard better on the right; on the left they were all short. Weber lateralized to the right.

No. 83.—Private C. B. Increased deafness, following the bursting of a shell twenty yards from him: previously deaf for years. There was a loss of membrana tympani in the left, and the membrane was red. The right membrana tympani was intact. Spontaneous horizontal nystagmus to right and left. The rotatory test to the left was normal; rotatory to the right, exaggerated response. Caloric to left normal; caloric to right, feeble response. All the forks were heard short on the left.

In the following cases the symptom giddiness was not complained of; two complained of feeling "dazed." In all a nystagmus was present. I regret in many instances I omitted to record whether a nystagmus was present. In "Experimental Physiology of the Semi-circular Canals in the Dog-fish,"[1] the following statement appears: "Pressure on an ampulla causes movements of the eyes and fins such as would occur normally if the animal's body were rotated in the plane of the canal stimulated." Consider also the rotatory test, which is mechanical stimulation of the ampulla.

No. 40.—Private F. F. Previously deaf, worse since he was blown up by trench mortar. Shrapnel wound of back. Feels dazed. Loss in right *nil*; ? laceration of left membrana tympani. There was horizontal nystagmus to right.

No. 41.—Private R. V. Shell burst on parapet. He was dazed; had discharge, pain and deafness on left; horizontal nystagmus to right. Tuning fork C_4 very short right and left.

No. 42.—Corporal L. Y. Trench mortar burst on top of the trench, causing deafness, noises in the left ear, and bleeding. . There is an intra-membranous clot at left umbo. Horizontal nystagmus right and left, more marked to right.

No. 43.—Private T. B. Shock due to the bursting of a prœmature. Caused deafness right and left and buzzing. There was rotatory nystagmus to the right. Tuning forks, not much loss.

No. 44.—Pioneer J. S. Always deaf; C.D.C. changes. Guns make him worse by bringing on buzzing noises which are much worse in left ear. Horizontal nystagmus to left.

[1] Lee, *Journ. Phys.*, 1903, xv, p. 328.

Analysis of Shell Concussion Effects on the Internal Ear.

The membrana tympani is of much greater area than that of the fenestra ovalis. The ossicles convey the vibrations from the large area to the small; much greater force must therefore be applied to the small area. We should expect different effects on the internal ear if the full force has been transmitted to the fenestra ovalis, the membrana tympani having withstood the force of concussion, and a less effect if the membrana tympani has been lacerated, much of the force being lost; and in cases of otitis media suppurativa with large losses, where the remains of the membrana tympani and the diseased condition of the contents of the middle ear have been much minimized in value as transmitters. I shall select cases falling into the three classes, and then consider the effects.

(*A*) *Membrana Tympani remained intact.*

No. 85.—K. Deafness, left most, right less; following Delville Wood; quite right before, never had discharge from ears. Has noises like "engines" in left ear now. No evidence of pre-existing disease.

	R.	L.
C_1	*Nil* A.C. or B.C. ...	*Nil* A.C. or B.C.
C_2	– short ...	*Nil*
C_4	Short ...	*Nil*
Rotatory test ...	Feeble and slow ...	*Nil*
Caloric test ...	Feeble ...	*Nil*
Weber	Weber	

No. 86.—B. Buried by shell; no previous trouble. Deafness, dizziness and noises followed.

	R.	L.
C_1	*Nil* ...	*Nil*
C_2	– short ...	*Nil*
C_4	Short ...	*Nil*
Rotatory test ...	Very slow, long excursion ...	Very slow, long excursion
Caloric test ...	*Nil* ...	*Nil*
Weber	Weber	

No. 73.—W. Was firing 12in. howitzer from Dainville Station; had been on large gun fire before. On this occasion the howitzer was in a cutting, two sides of which were closed in.

	R.	L.
C_1	+ ...	+
C_2	+ ...	+
C_4	Very short ...	Very short
Rotatory test ...	Normal ...	Normal
Caloric test ...	Normal ...	Normal
	Weber	

Vide Nos. 52 and 42 previously mentioned.

No. 64.—B. Following Mametz Wood. Deafness right and left, singing noises; previous C.D.C.

	R.	L.
C_1	+ short	+ short
C_2	+ short	+ short
C_4	Very short	Very short
Rotatory test ...	Normal	Normal
Caloric test ...	Normal	Normal

Weber does not lateralize.

No. 54.—B. Was buried. Deafness right and left at first, right passed off in seven or eight hours; was dumb for three hours; giddy and straining noises. No previous existing disease. There was a blood spot in the left superior quadrant.

	R.	L.
C_1	+	– short
C_2	+	Neutral
C_4	Good	Very short
Rotatory test ...	Normal	Normal
Caloric test ...	Normal	Normal

(*B*) *Membrana Tympani Lacerated.*

No. 49.—McG. Shell burst a few yards from him, injuring the left side of face, jaw, and eye, and causing deafness, giddiness, pain, buzzing, and discharge of blood in left ear. There was a laceration at left umbo and hæmorrhage in the attic region. Right intra-membranous clot at umbo and small blood spots.

	R.	L.
C_1	Neutral	–
C_2	–	– short
C_4	Heard well	Heard short
	Weber	
Rotatory test ...	Feeble	Feeble
Caloric test ...	Feeble	Feeble

No. 55.—F. Trench mortar fell and exploded on top of parapet; caused deafness right and left. No previous disease. There was a blood streak down the handle of the malleus on the left, and the membrana tympani was lacerated at the anterior inferior quadrant.

	R.	L.
C_1	–	–
C_2	– very short	– very short
C_4	Very short	Very short
Weber	*Nil*	*Nil*
Rotatory test ...	Feeble	Feeble
Caloric test ...	Feeble	Feeble

Vide No. 56 previously mentioned.

No. 59.—Lieutenant P. Trench mortar fell on trench and burst two yards from him. Pre-existing disease *nil*. Deafness, left. The left membrana

tympani was very red and lacerated at anterior inferior quadrant; discharge of muco-pus, blood-stained.

	R.	L.
C_1	Felt only	... Felt only
C_2	+	... – very short
C_4	Heard well	... Heard very short
Weber		... Weber
Rotatory test ...	Normal	... Very exaggerated normal
Caloric test ...	Not performed	... Not performed

Vide No. 60 previously mentioned.

No. 63.—M. A shell burst at the back of him and he was blown into the air. Deafness right and left, unconscious 1½ hours; continuous headache, hissing noises; right improved, not left. Pre-existing c.d.c. Left membrana tympani lacerated at inferior quadrant.

	R.	L.
C_1	+	... – short
C_2	+	... – short
C_4	Heard well	... Very short
Weber	Weber	
Rotatory test ...	Normal	... Feeble
Caloric test ...	Normal	... Feeble

No. 65.—P. Heavy guns, fired whilst he was in a communication trench, caused deafness and noises, and discharge from left ear due to laceration in upper quadrants.

	R.	L.
C_1	– very short	... – short
C_2	– very short	... – short
C_4	Very short	... Short
Weber	Weber	
Rotatory test ...	Feeble	... Feeble
Caloric test ...	Feeble	... Feeble

No. 74.—E. S. Big shell exploded a few feet off; wounds of hands followed by left deafness, blood from left, hissing noises, discharge two days later. There was general redness of left membrana tympani and a laceration at anterior inferior quadrant.

	R.	L.
C_1	+	... –
C_2	+	... – short
C_4	Normal	... Very short
Rotatory test ...	Exaggerated	... Feeble
Caloric test ...		

(*C*) *Membrana Tympani Previously Lost.*

No. 11.—S. Shell burst near him, 15 yards off. Always deaf right and left, deafness much increased by the explosion. ? congenital syphilis.

	R.	L.
C_2	*Nil*	... *Nil*
C_4	*Nil*	... *Nil*
Rotatory test ...	Normal	... Normal
Caloric test ...	Normal	... Normal

No. 23.—Previously mentioned.

No. 28.—B. Aërial torpedo burst on parados, buried him; deafness, right and left, noises, inability to walk. Right ear got better.

	R.	L.
C_2	+ short	... + short
Weber	Weber	
Bone conduction over left mastoid heard in right ear *only*.		
Rotatory test ...	Exaggerated	.. Exaggerated
Caloric test ...	Exaggerated	... Exaggerated

No. 47.—*Vide* previously mentioned.

No. 75.—B. Trench mortar shell burst and he was struck by mud; face and scalp injuries. Deafness in left, discharge after three days; singing in left, blood in throat. Pre-existing old losses right and left, recent slit in posterior superior quadrant, left, and a hæmorrhage in posterior superior quadrant, right.

	R.	L.
C_1	–	... – short
C_2	–	... – short
C_4	Heard well	... Short
Weber test ...	Weber	
Rotatory test ...	Normal	... Normal
Caloric test ...	Normal	... Feeble

No. 83.—*Vide* previously mentioned.

Class A: In only two cases (85 and 86) of the 100 examined was there total inability to hear tuning forks C_1, C_2, C_4; in these cases also there was practically no response to caloric tests, and there was peculiarity of the nystagmus to rotatory tests, showing some marked derangement. No. 85 required a full quart of *hot* water to produce reaction, which was then feeble and slow. No. 86 required nearly as much additional turning to produce a reaction which was then of extreme excursion, the eyes moving to their full limits—internally and externally—and very slow. Effect of explosion fairly equal on right and left, and C_4 most affected.

In Class B there was never total inability to hear C_1, C_2, and C_4, and only feeble response to rotatory and caloric tests, except in a couple of cases where rotatory and caloric tests gave much exaggerated response.

In Class C there was never total inability to hear C_1, C_2, and C_4, except in No. 11, and I am inclined to think he was a case of otitis interna syphilitica, deprived of his small amount of hearing by the explosion, and the response to rotatory and caloric tests was more

often normal than otherwise, although excitation and depression were seen.

Taking the cases collectively the forks C_1, C_2, C_4 are generally shortened on the side most affected, or equally on right and left if both have been equally exposed. The tone area for C_4 is certainly the most affected. There is at times marked shortening for C_1 and C_4 in comparison with C_2. Again, C_1 and C_2 may be normally heard and C_4 at very short distance. Weber lateralizes to the side least affected, and though both right and left were seriously disorganized in Nos. 85 and 86, there was lateralization to the side on which the forks were still faintly and shortly audible. In No. 73, Weber C_2 lateralized to a side—the sides having equal acuity. In No. 59, Weber lateralized to a side—very defective although the opposite side was normal. On performance of Weber test with the two forks C_1 and C_2—in some cases the former would lateralize to one side and the latter to the other, or C_1 would lateralize and C_2 not do so—and vice versa.

Caloric tests : Very much more water, either hot or cold, was required in performing the labyrinth tests as compared with amount usually required for functionating in normal cases. Two quarts and more had often to be used before a response was noticed. The nystagmus varied very much, but it was definitely present in every case except Nos. 85 and 86. It was often very quick and feeble, this was the common one—or very slow and feeble—in one case the nystagmus was so coarse in its movements, I can only compare them to the movements of the pendulum of a big clock, slow with long excursions. In a few cases the nystagmus was quickly induced and very violent. In these cases the patient looked deadly pale, with extreme giddiness, faintness, and vomiting ; in two the effect was so prolonged the cases were unable to return to their ward for fifteen to twenty minutes.

The rotatory and caloric tests corresponded in their effects except in a very few instances, notably No. 83.

It is evident that certain parts of the organ of Corti are often more seriously damaged than others, and that the labyrinth may be rendered hypo—or hyper—excitable. Anyway, its normal equilibrium is much altered. It is evident that most effect on the internal ear is produced through an intact membrane, and one which stands the force of the explosion ; also that the effect is less when the membrana tympani lacerates, and still less in the conditions of previous loss of membrana tympani.

Causation and Prognosis.

Shell concussion deafness is possibly due to three contributory factors:—

(1) Cerebral concussion.

(2) Overstrain and fatigue of the organ of Corti: the former being due to violent oscillations of the perilymph communicated to the organ of Corti, and the latter to continuous violent noises or explosions at close quarters.

(3) Temporary or permanent disorganization of the conductive apparatus.

The prognosis depends on the recovery of these parts.

(1) The patient experienced a great shock and was, in many instances, buried. He became suddenly deaf, and often dumb also. In other cases, he could not see, or had paresis of arm, leg, or both legs. Here the higher centres were temporarily involved. One watched the almost sudden improvement in hearing in a few days as shock passed off, and the disappearance of nervous symptoms generally when the patient was removed from the firing line.

(2) A portion of deafness remains to be more or less slowly recovered from by the return of the internal ear to a normal or nearly normal condition.

(3) Structural damage must leave a permanent imperfection of function—a ruptured membrane; a dislocation, partial or complete, of the small bones from one another, or the stretching of their attachments to the tympanic wall.

These, in other larger but similar conditions, are only slowly recovered from, if ever in many instances. I am able to quote from cases seen three or four months later, showing a great recovery of hearing. The loss in these cases was practically due to the macroscopic structural damage, and the frequent sequela, chronic otitis media suppurativa, but from the information gained from the cases I have seen, I believe that shell concussion deafness is, to a large extent, temporary and curable.

Major SHUTER.

I would like to make a few remarks on one aspect of the subject. My experience has been with cases of ear injury some months after it has been inflicted—i.e., in the Australian troops who came from Gallipoli, and from the English hospitals.

I shall not speak of direct traumatism. I am in accord with most of what has been said, but I wish to speak of what is termed concussion injury, and what a second speaker referred to as nerve deafness. I have seen a large number of such cases, but my work has not enabled me to go so thoroughly into the matter as the last speaker has gone: mine is a roving commission. I differentiate, however, between machine gunner's and concussion deafness; in the former there are islands of deafness. I have seen a large number of cases which show the following combination of signs. They frequently occur in those who have a very patent meatus, and, contrary to Lermoyez's opinion, I found them more frequent in those with pre-existing middle-ear disease. Mr. Scott asked: "How do you know they have had pre-existing middle-ear disease?" In these cases the obvious signs of middle-ear disease are so frequent that, when one considers the number of normal middle ears compared with diseased middle ears, it is a reasonable opinion to state. They are due not to sound waves, but to the wind waves caused by a shell explosion. I have seen them also in a case in which a German mine was exploded in a confined space; I have seen them in trench cases and after bomb explosions, in which the patient has been rendered unconscious and deaf for forty-eight hours. They present themselves some months afterwards with this combination of signs: marked diminution of bone conduction, tinnitus, paracusis, hearing better in a quiet room, and hearing lower tones by air conduction proportionately better than higher.[1] Yet apparently they have a very complete range of hearing. I should like to hear from some present why we assume that diminished bone conduction and diminished air conduction for the higher forks means nerve disease. I do not know where the statement originated, or on what reasoning it is based. One can physiologically alter the bone conduction by putting the finger in the ear, or by inflating the ear, or

[1] The range of hearing was taken by a monochord, which shows perception of pitch up to an average of 25 cm. by air conduction, and 18 to 20 cm. by bone conduction. This is only a little lower in my experience than a normal ear.

by the Gelle test. It is altered very much by middle-ear disease, and a very pertinent fact is that it is greatly increased in cases where there is fixation of the stapes. I think in the cases I refer to there is some alteration in the conducting apparatus—it may be the ossicles, it may be the foot-plate of the stapes, or an alteration in the accommodating mechanism of the opposing action of the stapedius muscle and the tensor tympani. However, the matter has puzzled me, and I should like to know whether others have noted this combination of signs. I should welcome any expression of opinion.

Mr. Sydney Scott.

It is highly significant that the Dominion Governments should have sent representatives of otology to investigate the ear cases among their Forces in the Army. Our methods of practice in otology in civil life singularly well prepare us for investigating cases we meet with in warfare. After a little experience of the actual conditions near the fighting line, at the base, and in England, I am convinced there is much for otologists to do. Certain cases of injury require considerable time to ascertain the true state of affairs. It is not sufficient merely to look at the drum and see how far a watch can be heard! Some cases will take an hour or more to examine. We need not only to apply tuning-fork and other hearing tests, but frequently have to investigate the vestibular functions, to say nothing of the examination of the nose, pharynx and Eustachian tubes. And the results are well worth the same care we take in civil life. With regard to the hearing tests there are three features I think we should consider. The first is the determination of the "tone-range"; the second of "tone-acuity," and the third "tone-analysis." The first is measured by forks and the monochord, which is far superior to Galton's whistle.[1] "Tone-acuity," for a given tone or group of tones of known intensity is recorded by the distance-limit of hearing a watch or acoumeter. Under "tone-analysis" will come the faculty of hearing the voice as whisper or in conversation. We have met with patients who have a short tone-range—e.g., 16 d.v. to about 1,000 d.v. or less, who can hear conversation at 2 or 3 metres. Others, whose hearing became defective after being buried by shell fire, may retain normal tone-range and have very defective tone-analysis —that is, they exhibit great difficulty in distinguishing words of speech,

[1] "Discussion on Value and Significance of Hearing Tests," *Proc. Roy. Soc. Med.*, 1912, v (Sect. Otol.), pp. 113, 131.

especially in a noise. We meet with similar conditions in civil life, but they have been classed under senile or nerve deafness of doubtful cause.

The importance of considering the vestibular system is self-evident to those familiar with the manifestations which injury to the ear frequently evokes. But it hardly seems sufficient merely to say that the caloric test was done with hot or cold water, without observing precisely what the posture of the head was during the application of the test; for otherwise we may obtain normal reactions on both sides, with the head in certain positions, though a unilateral lesion actually exists. As an example, in a man suspected of malingering, I found by setting his head in requisite positions that one of his semicircular canals did not react to the caloric tests, whereas all the other five canals reacted normally. The results of "caloric" and "rotation" tests tallied with each other. The man had no appreciation for the short monochord in the affected ear (i.e., high limit was below 5,000 d.v.), but he could hear my long monochord. The low tone limit was raised on both sides. He had active nasopharyngeal catarrh at the time, and catheterization definitely improved his hearing for low tones, bringing him back to the 16 d.v. limit, though it did not of course affect the high tone loss. Nevertheless the hearing became good and useful, both as regards tone acuity and tone analysis.

We all greatly appreciate the very fine work Mr. Fraser has sent us to-night. We see there one end of the story in the pathological findings in fatal cases subjected to the effects of high explosive violence, but we have yet to see the concurrent clinical manifestations recorded side by side with such pathological findings. In the specimens which I showed last year[1] from the soldier who was wounded in the vertex of the skull, there was free hæmorrhage in the tympana, without fracture or rupture of the drum or any other injury such as we see in some of Mr. Fraser's cases, and although, fortunately, it was possible to test my case during life, the records were incomplete because suitable instruments were not available at the time. We hope this gap in our knowledge will soon be filled, as the result of such work as it is now possible to carry out. I think, Sir, we might commend to the War Office the appointment of more otologists to investigate and report upon the various forms of injury to the ear, due to the War, in the way that is being done in the Canadian hospitals for the sake both of the individual and the State.

[1] "Histological Preparations of the Labyrinths and Tympana of a Soldier suffering from Deafness due to a Bullet Wound of the Vertex of the Skull," *Proceedings*, 1916, ix (Sect. Otol.), p. 28.

Captain E. A. PETERS, R.A.M.C.

Deafness due to gunshot wound and high explosive concussion falls into three groups:—

(1) (*a*) The membrana tympani may be ruptured by gunshot wound or high explosive concussion; (*b*) the auditory meatus obstructed by a fragment of metal; (*c*) the middle ear may be filled with blood. The smaller extravasations are absorbed, but others break down into abscesses. The condition may occur as the result of the regional disturbance or high explosive concussion.

(2) Labyrinthine deafness. This is probably due to hæmorrhage or vasomotor change. It is a very common form of war deafness and the prognosis does not seem to be good. I have not been able to observe the microscopical condition, but that such a condition exists may be assumed (*a*) from the œdematous congested state of the larynx observed after gunshot wounds in the proximity; (*b*) the hæmorrhage common in the brain with intact dura mater consequent on gunshot wound fractures in the vicinity. It results from direct blows or high explosive air-conducted concussion. The following is a typical case:—

M. Buried by a shell fourteen days previously; he was hit by a fragment behind the right ear.

0·		S. watch		$\frac{24}{30}$
– 15 seconds	...	c mastoid	...	Normal
18,000	...	Monochord	...	30,000
T. F. 64	...	Lower limit	...	T. F. 64

Rotation for external semicircular canal, clockwise: exaggerated nystagmus; anticlock, normal.

There was some evidence of fatigue, but no giddiness.

The patient is usually concussed, and the ear exposed to the fuller force is more affected. The semicircular canal system and cochlea may be affected in different degrees; the cochlea does not recover so quickly as the semicircular canals. Both ends of the register are affected, but the higher notes more regularly.

(3) Psychical or central deafness is comparatively rare. It is symmetrical and usually absolute deafness, and is often associated with mutism or stuttering speech. This form of deafness is dealt with by Major Hurst.

These three forms of deafness may coexist and contribute a factor in different proportions in a given case.

Major A. F. Hurst, R.A.M.C.

As a result of my observations on cases of functional deafness, deaf-mutism and so-called "hyperacusis" in soldiers in the Neurological Section at Netley, I have come to certain conclusions as to their ætiology.

(1) *Deafness.*—The deafness is hysterical and due to auto-suggestion. The momentary deafness, which is the natural result of the terrific noise caused by the explosion of a big shell in the immediate neighbourhood, may make such an impression on the mind of a soldier that, on coming to himself, whether he has actually lost consciousness or not, his first thought is for his hearing, especially if it was already impaired by preceding disease, and he may be so convinced that he is permanently deafened that he becomes actually deaf as a result of auto-suggestion. Hearing necessitates listening. Inattention during a dull sermon results in total deafness to the sermon, and in hysterical deafness the patient is so convinced that he cannot hear that he does not listen; although the sound vibrations reach the ear in the normal way, they do not give rise to the slightest auditory sensation because of this inattention. The synapses at one or more of the cell-stations in the auditory path to the cerebral cortex must therefore be unswitched, probably as a result of retraction of the dendrons. I shall presently show that the auditory-motor or "jump" reflex is a function of the mid-brain. In the absolute deafness of shell shock this reflex is abolished or greatly diminished. A very nervous but totally deaf mute remained completely unmoved, never jumping nor showing a flicker of his eyelid during one of the severest thunderstorms I have known, and yet the next day he was completely cured by suggestion. In some cases the reflex is present when first noted, but disappears more or less completely when the test is repeated. One of the unswitched synapses must therefore be below the mid-brain, and either in the auditory nucleus or, less probably, in one of the intermediate cell-stations—the superior olive or the nucleus of the lateral fillet. It is very much easier to cure pure mutism than pure hysterical deafness, because by simple persuasion and re-education the mute can be induced to use the muscles involved in speech, and in severer cases the excitement caused by some unexpected event, or by alcohol or ether, is enough to cause involuntary speech, for when once a word has been uttered involuntarily the mute is convinced that he is cured. If this occurs in

a deaf-mute, the return of speech generally convinces him that he can also hear; he therefore listens, and the unswitched synapsis is once more switched on. Deafness without mutism in shell shock is, in my opinion, almost always of the same nature and not due to any gross lesion of the labyrinth, as so far as I know this has never yet been proved to occur in the absence of external injury; the ruptured drum often seen is also of small importance, as it in no way influences the rapidity of recovery in hysterical deaf-mutism. In spite of this it is often exceedingly difficult to restore the hearing in the absence of mutism, and many cases have, I believe, been regarded as the result of some actual destructive lesion because of their persistence. In such cases suggestion under hypnosis almost always cures unless the deafness is so absolute that the patient cannot even hear shouting through an ear-trumpet, in which case it is impossible for suggestions to have any effect, as the unswitched synapsis remains unswitched in hypnosis, and commands, which in other hypnotized individuals are immediately obeyed, do not reach the cortical centre of hearing and are therefore not obeyed. As I have found that hysterical manifestations, such as contractures and anæsthesia, disappear during sleep, it might be expected that hysterical deafness would behave in the same way, but this is not the case. Three men suffering from total hysterical deafness without other symptoms were put into a hut by themselves. During the night an alarm of fire was raised, but in spite of shouting and banging a poker against a shovel within a few inches of their ears, neither of the two who were asleep when the ward was entered woke up. The auditory motor reflex occurred in one patient, in whom it was present when awake, but in the other, who had no such reflex when awake, it was absent.

(2) *Exaggerated Auditory Motor or Jump Reflex, so-called Hyperacusis.*—A sudden noise normally causes an individual to jump and blink, and at the same time his pupils dilate; the "jump," at any rate, is a protective reflex and represents the preparation for flight or fight. An officer whose left motor cortex had been almost completely destroyed went to see "The Man that Stayed at Home" about four months after he was wounded. His right arm jumped violently when the gunshot rang out on the stage, although no trace of voluntary movement returned until three months later. The motor part of the reflex is thus subcortical. In war neuroses in which the reflex is exaggerated, jumping continues during sleep and deep hypnosis, although the patient does not hear the noise which induces it, even

in a dream. That the reflex is quite independent of actual hearing is shown by the fact that during deep hypnosis with the eyes open, individuals in whom the reflex is normal or only slightly exaggerated do not jump or blink, and the pupils do not dilate even when a poker is banged against a shovel within a foot of the ear, although they answer even whispered questions and obey whispered commands.

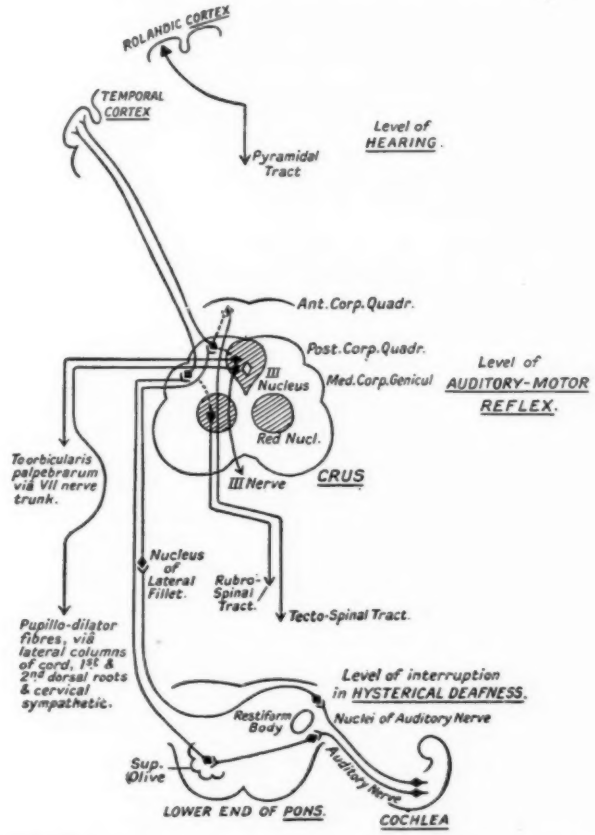

Diagram to illustrate the neurones concerned in hysterical deafness and in the auditory-motor reflex.

I have already pointed out that the reflex is abolished in complete hysterical deafness, so it must occur above the synapsis, which is unswitched in this condition. This suggests that the reflex centre is

in the posterior corpus quadrigeminum or medial corpus geniculatum, and the close relation of these centres to the blinking and the sympathetic pupillo-dilator centre in the neighbourhood of the third nerve nucleus and to the anterior corpus quadrigeminum and red nucleus, in each of which a descending motor track originates, gives an anatomical basis for this view[1] (*see* fig. p. 117). The very common exaggeration of this reflex in soldiers suffering from certain war-neuroses is not, therefore, correctly described as hyperacusis, for the sense of hearing may be no sharper than normal, and actual hearing need not occur at all, as the reflex occurs when they are asleep and hypnotized; it is simply a part of the general exaggeration of the defensive reflexes, which is a very characteristic feature of those war-neuroses that are due rather to emotions than to the actual concussion of pure shell shock. In severe cases the patient appears to be in extreme terror: he jumps violently and trembles from head to foot at the slightest sound, and raises his arm as if to protect his face from a blow and hides his head under the bedclothes when anybody approaches him. Terror is at first actually present, for the patient never ceases to think of the horrors he has passed through, and he constantly pictures them before him, while every sound reminds him of the bursting of shells. But after all trace of terror has disappeared as a result of suggestion under hypnosis, and the patient no longer thinks or dreams of the horrors of war, the appearance of terror may continue unabated. This is due to jumping and flinching being defensive reflexes; the intense emotions to which the patient has been subjected leads to such an increase in the excitability of the central nervous system that the exaggerated reflexes persist after the cause of their exaggeration has disappeared. In one severe case true hyperacusis was also present, and Captain Peters estimated that the patient heard sixteen times more acutely than the average normal individual. The hyperacusis and exaggerated defence reflexes were entirely uninfluenced by the administration of 100 gr. of bromide a day. The possibility of the jump reflex occurring in the absence of actual hearing, and being absent when hearing is not abolished, is of some practical importance, as I understand that the presence or absence of the reflex is often used as a test of complete deafness in mute children and in supposed malingerers, whereas its presence is not really a proof that actual hearing occurs.

[1] Since this note was written Professor C. S. Sherrington tells me that he and Forbes found that an incomplete jump reflex occurs in the decerebrated cat, and that both the posterior corpus quadrigeminum and medial corpus geniculatum were concerned in the reflex.

Mr. E. D. D. Davis.

My experience agrees with what you have said, Sir. But there is one question I would like to ask Dr. Gordon Wilson—namely, the difference between nystagmus of cerebellar injury, and that due to injury of the labyrinth.

Lieutenant-Colonel Goldsmith, C.A.M.C.

I shall address myself to the question asked by Dr. Grant, as to the amount of hearing necessary for a man to be in the Service, and the question of middle-ear suppuration. It is laid down in the Army Council instructions what amount of sight a recruit must have in order to be considered fit. That standard has been lowered as man-power has become more necessary. But the amount of acuity of hearing necessary has not been laid down, nor is it possible to lay down in an accurate way. The Canadian Army Medical Service has tried to lay down how far off a man should hear conversation voice in order for him to be placed in A, B, or C classes respectively. A man who hears ordinary conversation 15 ft. away in each ear, and who has no organic disease of the ear, can be accounted fit for Class A. But it must be known with certainty that the other ear has been carefully closed during the test and that the speaker has not been seen, because many men in the course of years have learned to get along by lip-reading. If a man who is at all deaf is put in a listening post, he may seriously endanger his company. If he can hear a conversation at 21 ft. with one ear and is deaf in the other, he is also classed A. The Canadian regulations are as follows:—

Hearing and Nasal Condition.—Category "A": A soldier who can hear 15 ft. or better in each ear by ordinary voice, and who has no organic disease of the ear, shall be placed in Category "A." A soldier who has hearing of 21 ft. in either ear, but with little or no hearing in the other ear, but without active organic disease, shall be placed in Category "A." No soldier with a discharge from his ears shall be placed in Category "A."

Category "B": A soldier who has hearing of 15 ft. in either ear and little or no hearing in the other ear, and has no active disease in either ear, will be placed in Category "B" (i). A soldier who has better hearing than 12 ft. in each ear, but has a discharge from either ear of a moderate degree, or is subject to frequent recurrent discharge from the ears, of a moderate degree, may be placed in Category "B."

Category "C": Soldiers who show symptoms of chronic ear conditions, such as hearing reduced to less than 15 ft., with freely discharging ear or ears, or shell concussion deafness, shall be placed in Category "C." A soldier who has fully and recently recovered from a mastoid operation shall be placed in Category "C" for three months and then regraded.

Category "E": Cases that show repeated signs of recurring serious ear conditions, such as recurring mastoid pain, free formation of granulation tissue, indications of bone disease, or free, full and offensive discharge from the ears, should be reported upon by an otologist with a view to being placed in Category "E" for discharge as permanently unfit.

Nasal Condition.—Soldiers suffering from severe nasal obstruction should be sent to a specialist medical officer in charge of ear, nose and throat diseases attached to one of the Canadian Military Hospitals, for report, and if necessary, treatment, before being sent overseas, as it is found that such cases find it difficult, and in some cases impossible, to wear respirators.

Tonsils.—Enlarged tonsils which were present previously to enlistment should not be removed unless they show signs of chronic inflammation, or recurring acute attacks of inflammation interfering with Active Military Service. All cases other than above, should be placed in Category "A." Where the removal of chronically inflamed tonsils will fit a soldier for Category "A," operation should be performed.

With regard to middle-ear suppuration, it is laid down in the Canadian Services—and I do not agree with it—that a man with middle-ear suppuration is not to be sent to France. Because a man has had discharge from his ear, it does not follow that he will "go sick" with it, and it will not do to say he is liable to mastoiditis or brain abscess, or acute suppurative labyrinthitis, since he is much more liable to be shot. A man whose hearing allows him to "carry on," and who has no active inflammatory condition in any of the surrounding structures, such as the labyrinth, the mastoid, or lateral sinus, can quite well go to France. And if you were now to bring from France all the men with chronic middle-ear suppuration, you would have battalions of them all over the country. In Boulogne, in 1914, I removed polypi which projected from the meatus, from dozens of men, and sent them back to the front. They had some discharge from the ear, but it was able to escape, and the men continued to be just as good soldiers. As the demand for men becomes greater, aurists should not put greater difficulties in obtaining men in the way of the military authorities. A man may have middle-ear suppuration for years without it causing him trouble, and the Regimental Medical Officer should not allow him to make use of it as an excuse to be sent back.

So far as concussion is concerned, it has struck me, on examining many of these cases, particularly for medical boards, that if you go into the history you will find many of the men have had deafness before the War, and their drums will show evidences of it: and particularly in that type of chronic middle-ear deafness with open Eustachian tubes and thickened drums, in patients who hear better in a noise but are not of the type called pure otosclerotics. These matters will be of great importance when dealing with pensions boards. If a man has been deaf before the War, and has been a lip-reader, that fact should be stated in reports. The amount of disability is based upon those reports, and it is very necessary that the aural report should be complete. If it says a man can hear ordinary conversation at 11 ft. with one ear, the word used in the examination should be stated, because when the case comes for review from the standpoint of a pension, someone else will examine him, and the same word should be used, otherwise the patient may say he is worse. I think Politzer's acoumeter should be used in all these cases. At the present time these matters are carelessly recorded.

In recording the tuning-fork tests, which I think should be done, particularly in pensionable cases, so that a record for future comparison may be obtained, it is necessary when stating the result of the Rinne test to do so with two forks, C 512 and C 256, and to record how many seconds plus or minus. Furthermore it is of value to record the low and the high limit. The high limit should be stated not by naming the highest fork heard, since this will probably not be more than C 3,012, but by recording the monochord reading, which definitely registers the upper limit. In special cases labyrinthine tests will be required, and even a neurological examination. Schwabach's test should be similarly recorded, and it is to be remembered that in cases of neurasthenic deafness the fatigue period is particularly noticeable. Many cases of perceptive deafness hear all the high forks but for a shortened period, and it will add to the accuracy of the report if this record is made.

Experienced aural surgeons should make the special reports on ear cases just as ophthalmic surgeons are so required to do in Chelsea Board cases, so that there will be a definite record of the man's condition. This will be invaluable to those who have to report a second time, when the pensioner comes up for review, and will ensure that while the State is more carefully protected, the man suffers no injustice.

One should be very guarded in advising nose and throat operations during war time. I am sure a great many officers and men have undergone operative treatment for conditions which have existed before

the War, and in whom active service has produced some increase of discomfort or inconvenience, but not enough for their state to be classed as a *disability*. The real disability is the operation itself, which results in the State losing the services of the soldier for many weeks. My own opinion regarding us Canadians is that we do not react very well, after nose and throat operations, in this country unless we have been here a considerable time. Our soldiers are all subject to an increase in their rhinitis and rhino-pharyngitis, which will subside in warmer weather. I have seen many cases of officers and men who have returned from France with various wounds, whose stay in hospital has enabled a long-standing nasal condition to be discovered, and who have undergone operative treatment even though no disability has been present. In civil life we are quite justified in relieving discomfort and inconveniences in our patients if they wish for such relief, but in military life the services of the soldier belong to the State and we should be very careful that we do not take too much liberty with such services. Furthermore I wish to draw your attention to the responsibility each one of us assumes when answering the question as to whether the disability is the result of active service, or whether it is aggravated by it.

Mr. Hunter Tod.

My experience of these cases, although not so great as that of others, as I am not attached to a military hospital, has been that bone disease, the result of direct injury by bullet or shell fragments, is more difficult to cure than in civil cases because of its more septic nature.

Partial nerve deafness and tinnitus, unaccompanied by any lesion of the tympanic membrane, is not uncommonly caused by the explosion of shells or from a projectile passing close to the ear. This was well shown in one case, that of a sailor, in which the tragus was taken off and the concha pierced by a bullet which just grazed the side of the head. In association with the traumatic perforations, tinnitus seems to be the chief subjective symptom, especially in regard to prognosis. What answer can we give to the repeated question: "When will the noises cease?"

I am interested in what has been said about the caloric test. It is a very important test with regard to prognosis in internal ear concussion, and therefore also from the point of view of pensions. I had an

officer who came to me shortly after the beginning of the War who was completely deaf for all sounds—the result of shell concussion—and he had been so for three months. He took a long time to react to the caloric test; but as he gave a mild reaction I told him, although I then had no experience to guide me, that he would probably recover his hearing. He went to Scotland, and I understand that his hearing is now practically normal.

In answer to Dr. Dundas Grant, there is no standard of hearing for our Army. I once applied for a definite statement as to the standard of hearing required for recruits, but the only answer I received was that the hearing should be good.

In cases of middle-ear suppuration we surely should take the same sensible view as in civil practice—for example, when testing for insurance. If there is perforation of the membrane, but no sign of bone disease, there is no harm in sending a man out to the front. If there is bone disease of the tympanic cavity or its walls, or signs of chronic mastoid disease, I would not send him out, because even if the soldier has no serious symptoms at the time of examination he will be a nuisance to the medical men at the front.

I take advantage of this meeting to suggest that we might combine in representing to the Army Council that we should be better represented in the Army, and that aural consultants should be appointed.

Mr. Somerville Hastings.

Since the beginning of the War, I have seen a good many cases of injury at two fair-sized hospitals, and what has struck me most is, the amount of injury the normal ear will stand without permanent affection of the hearing power. I have seen comparatively few cases in which I could be sure the membrana tympani had been recently ruptured. Very many cases of perforation have come under my notice in which the deafness was stated to have followed a recent injury, but in only a few of these did the appearances agree with this. I have also noticed the frequency with which both types of concussion deafness occur in ears already injured by perforations without discharge, or by thickening of the drum, or scarring. Another point which has not been mentioned to-night is, I think, the frequency of cases of suppuration in which one gets a history that when the patient was a child, suppuration in the ear occurred for a time, or in which there may be a large perforation with

no history of suppuration at all. And yet suppuration starts again after a concussion, or after the man has been blown up by a shell, or buried in a trench.

The PRESIDENT.

In the interesting discussion we have had many important points have been brought forward. I do not profess to be able to answer the questions which have been asked, as I have not had sufficient experience, my main work in connexion with the wounded having been general surgery. This discussion has opened up fresh points for us to think over and to bear in mind when examining patients in the future.

Section of Otology.

President—H. J. Marriage, F.R.C.S., Captain R.A.M.C.

(*May* 18, 1917.)

Cerebrospinal Fluid escaping from the Ear for Twelve Months.

By J. F. O'Malley, F.R.C.S.

Driver E. R., aged 27, came under my care seven months ago, with the following history: He stated that he underwent a mastoid operation, performed by Dr. Paterson, of Cardiff, about ten and a half years ago. The result was excellent for about six years, when a polypus formed and was removed. After this he emigrated to Australia, and during his two years' residence there he had polypi removed twice. About eighteen months ago he had a polypus removed at Nottingham, and the ear began to drip soon after, the escape of fluid being worse at night.

When he came under my care early in September, 1916, the condition of his ear was as follows: There was a free flow of watery fluid dripping from the lobule of the right ear and escaping from the external meatus. The meatus was completely blocked by a smooth cyst-like swelling, which, if examined hurriedly, would be easily mistaken for a polypus. It was the scar tissue lining of the mastoid cavity, raised by the cerebrospinal fluid from the bony facial ridge and antral cavity. There was some pus in the meatus also. After keeping him under observation for about a week and getting the meatus as free from pus as possible, I decided to re-open the mastoid and clear out the old scar tissue and the focus which provided the pus. In doing so no difficulty was encountered, but I was unable to localize the bony defect, though I formed the opinion then that it was in the promontory. The operation

cavity was packed with sterile gauze soaked in iodoform emulsion and the usual dressings applied. These were all soaked through next day by the cerebrospinal fluid and had to be replaced. The same condition was found on the second day and also on the third, but as he complained of discomfort all dressings were removed, when the mastoid skin wound and the whole operation cavity was found profusely bathed in pus.

The cavity and wound edges were now cleaned twice daily with 40 per cent. rectified spirit and loosely packed with ribbon gauze wrung out of this solution. A material improvement set in in a few days, but the cerebrospinal fluid continued to soak through the dressings for six weeks. As the cavity was now almost free from pus, I dusted in a little boric powder and packed it very firmly with sterile gauze each day. I was now able to localize the point from which the fluid escaped very definitely—namely, the posterior part of the attic, near the aditus. The escape of fluid gradually diminished, but even as late as December 28, 1916, that is, about three months after operation, 1½ dr. was collected in four hours.

The leakage of cerebrospinal fluid ceased early in January, 1917, and the mastoid wound and middle-ear cavity were soundly healed before this date, but the antral cavity has been slow to fill up and form a strong scar. It is still supported by a gauze packing inserted twice weekly, although the soldier has returned to light duty and is otherwise well.

The degree of hearing in this ear is negligible, but the labyrinth responds to caloric tests.

The case seems to be of special interest for the following reasons:—

(1) The large amount of cerebrospinal fluid which a patient may lose over a long period without any apparent injury to health. I have estimated that he lost over 1½ gallons in three months.

(2) The influence of the cerebrospinal fluid in producing profuse suppuration in the mastoid cavity after operation.

DISCUSSION.

Mr. C. E. WEST: This is an extraordinary case, both as to duration and the absence of any symptoms referable to the escape of the fluid, seeing the enormous total quantity of fluid which must have been lost. I have never seen a parallel case in which escape took place from that situation; but one is familiar with escapes of fluid through the internal auditory meatus after translabyrinthine drainage, and with cases of escape both from occipital

drainage and after meningitis. Recently I have had two escapes from the lateral ventricle, but in none of them has the flow persisted beyond three weeks. It is difficult to see what could have been the cause of the prolonged escape in Mr. O'Malley's case. Was there any evidence of raised cerebrospinal pressure? Apart from that, I can only conclude there was something in the nature of what ophthalmologists call a "filtration scar." One can scarcely conceive of there being a hole unfilled by tissue for a period of twelve months. Did the patient have headaches?

Mr. SYDNEY SCOTT: I am reminded of a similar condition in a girl whose right labyrinth I opened eleven years ago. Two or three years later she came to the out-patient room saying that on several occasions she had noticed "water" dripping from the ear. I found that fluid having the chemical characteristics of cerebrospinal fluid dripped from her ear for about three days at a time. As it recurred I explored the mastoid cavity and discovered the fluid escaping, not from the labyrinth, but through the planes of dura mater of the middle cranial fossa, which had been exposed at previous operations. Soon afterwards the flow stopped spontaneously, and had not returned when I saw the patient for the last time three or four years ago. These cases present certain features in common with those of cerebrospinal rhinorrhœa which Sir StClair Thomson has described and collected. A few years ago a man was under my observation at the National Hospital for the Paralysed and Epileptic suffering from violent headaches, which were relieved by the spontaneous escape of cerebrospinal fluid from the nose. This recurred repeatedly for over a year. I think Mr. O'Malley's case probably belongs to the same category.

Mr. RICHARD LAKE: The first time I removed a cochlea I took away the modiolus, and there was a free flow of cerebrospinal fluid. As a result there was a great deal of irritation of the skin. I closed up the hole, after a fortnight, with a plug of wax. I wondered whether that might have any bearing on the cause of suppuration in the ear—namely, the irritation from this fluid. One would have thought the fluid was non-irritant.

Mr. H. BANKS DAVIS: One point which interested me very much was this. The note says: "The external meatus was completely blocked by a smooth cyst-like swelling, which, if examined hurriedly, would be easily mistaken for a polypus." A child I operated upon at the hospital four years ago, went into one of the other departments. From there she was referred back to the aural department with the statement that she had a polypoid cyst at the back of the ear, and a request that it be removed. I did not see the case until after an attempt had been made to remove it in the out-patient department by means of the snare. They opened a cerebral hernia. It was scraped, and the child sent home. She was brought back unconscious a week afterwards, with a temporo-sphenoidal abscess and an abscess of the cerebellum. The child is now quite well, but only as a result of extensive operations. I think the

posterior wall had been removed, and the brain had been sagging down into the meatus. I always think the removal of polypi in the out-patient room should be undertaken with very great caution.

Mr. O'MALLEY (in reply): I felt that I was dealing with a touchy condition, and I feared the onset of meningitis. My conclusion—if one can draw a conclusion from one case—is that the question of meningitis rests very largely on drainage. I did not begin the packing until I felt sure there was a considerable amount of granulation material. One should not immediately attempt to do firm packing in order to arrest the escape of the fluid; we should wait until there is a good covering of granulation tissue. If packing is then done, it provides a protection against infection. With regard to the profuse suppuration in the mastoid cavity after operation, it seemed very extraordinary to me, on opening the mastoid, to find pus pouring from the cavity. But nothing happened, and the man is now in far better health than at any period during the persistence of the discharge. Formerly he complained of headache and a languid feeling, but has not done so once since the flow has been completely arrested. Apparently, therefore, there is now no persistent pressure. The margins of the meatus look a little thin, and one might have supposed there was fluid near the surface, but I tested for that, and found none. To be quite sure, however, I still pack twice a week.

(*May* 18, 1917.)

Objective and Subjective Ticking in the Ears.

By W. H. JEWELL, M.D.

V. G., A GIRL, aged 11. The ticking began in the ears three months ago and could be heard at three yards distance, but it is not so loud now. It ceases during sleep, swallowing, or deep pressure behind the angle of the inferior maxilla, is independent of the pulse-rate, but corresponds in number and is synchronous with the clonic contractions of the tensors and levators of the soft palate, which are 150 per minute. There is neither blepharospasm, nor any movement in the tympanic membranes, corresponding to the ticking discernible.

Mr. JEWELL: I think this is a case of reflex neurosis, probably due to the presence of adenoids in a child of a neuropathic disposition.

(*May* 18, 1917.)

Case of Squamous-celled Carcinoma of the External Auditory Meatus and Tympanum in a Young Woman aged 22.

By Sydney Scott, M.S.

The following is an account of a case of malignant disease which had extensively destroyed the external osseous meatus, while a portion of the growth had invaded the tympanic cavity without destroying its bony walls or ossicles. The patient was first seen on January 26, 1917, on account of deafness and purulent discharge from the right ear. The meatus was full of what looked like granulations and a band of scar tissue which bisected the meatus in the region of the isthmus. She stated she had had intermittent otorrhœa for many years, and that it had been continuous for the previous twelve months. A small swelling formed immediately below the meatus and developed into a subcutaneous abscess, which was opened on February 1. She had no pain, nor tenderness, nor facial paralysis, nor pyrexia. The deafness was moderate and of the ordinary middle-ear type. Slight dizziness had been complained of, but the labyrinthine reflexes were normal. The nose, pharynx, left ear, and cervical lymphatic glands were unaffected.

On February 8 the inframeatal sinus, formed by the opening of the abscess a week previously, admitted a probe upwards and inwards as far as the tympanum, as the subsequent operation proved, without encountering bony resistance. The patient agreed to submit to an exploratory operation, and on February 15 the mastoid process was opened and found to contain normal air cells throughout. The meatus was then opened from behind and a tough troublesome bleeding growth, which had spread through the floor of the meatus into the inframeatal tissues of the neck, was removed. The tympanic plate had been destroyed together with the lower part of the tympanic membrane, the upper half of which was intact, with the handle of the malleus projecting from it. The antrum was then opened and seen to contain more of the growth, which here looked like a polypus projecting backwards, but not completely filling its cavity. The incus was seen to be embedded in, but not destroyed, by the growth.

The operation was completed a week later, that is on February 23,

after the histological examination had proved that the disease was carcinoma (Professor F. W. Andrewes). The limbs of the post-aural incision were extended to the temporal region and to the angle of the jaw, and the pinna, with the surrounding skin, was reflected forwards over the cheek. The whole of the mastoid process was removed, together with the fibro-cartilaginous meatus and concha, and all parts down to and including the tympanic ring, outer attic wall, tympanic membrane, auditory ossicles, and remains of the growth which occupied the tympanum. The stapes was left in position, likewise the labyrinth and facial nerve. Only a portion of the parotid salivary gland was removed with the main pre-auricular lymphatic gland, although this was not found to be involved. The wound was closed by suture in the usual way, and healing has taken place uninterruptedly since, but without the insertion of packing or skin-grafts.

No operation for the removal of the cervical lymphatic glands has yet taken place, for it is difficult to get the patient to realize that any further operation would be likely to prolong her life or add to her future comfort.

Comment.—Apart from the patient's youthful age for carcinoma of the ear, the following features appear to be noteworthy:—

(1) The total absence of pain while the disease was active.

(2) The apparently satisfactory extirpation of the growth.

(3) Its evident origin in the meatus.

(4) The question of the absolute necessity or otherwise, of removing the lymphatic glands before they become obviously involved.

(5) The prognosis in comparison with other cases of carcinoma of the ear.

DISCUSSION.

Mr. C. E. WEST: Carcinomata of the meatus represent a very much larger proportion of the total number of carcinomata which are reported as carcinomata of the middle ear than most people admit. That is a point upon which I have insisted on a previous occasion. The cases all have a strong family likeness. Most of them originate in the floor of the meatus, invade the tympanum, and penetrate downwards into the neck and towards the root of the styloid process. They are locally malignant. In my experience, they have rarely had any wide distribution as glandular involvement, and, generally, they recur. (I do not know about Mr. Sydney Scott's results, which, I believe, are better than mine.) The recurrence takes place along the line of the Eustachian tube and the base of the skull, and the patient dies of infection, or meningitis, or acute encephalitis. In some cases the patient has complete freedom for a long time—sometimes permanently—after a sufficiently bold operation.

I have known a patient, after two years of freedom from disease, come back and die from recurrence. I also know a patient who has survived for nine years, and passed through various accidents, and yet had no recurrence of his carcinoma. One patient must now be very old, if he is still living—and I have not heard of his death. I was interested to find Mr. Scott reporting this case, because only a week last Wednesday I operated on a case of squamous-celled carcinoma of the external auditory meatus at St. Bartholomew's Hospital, and it was unlike anything I have seen before. The patient, a woman aged 54, came up a fortnight before the operation, complaining of trouble, apparently furuncular, in the external auditory meatus. I am afraid that, in the press of out-patient department work, I did not go further with it: I turned the case over to the house surgeon, who incised it. A week later the woman returned with the meatus full of a fungating growth. There was a fixed mass, about the size of a filbert nut, below the meatus. The whole of the floor of the anterior wall of the bony meatus had been destroyed, and the growth in the meatus was continuous with that in the neck, where it went downwards $\frac{3}{4}$ in., and inwards practically as far as the styloid process. It had invaded the tympanum, but only, I think, in a polypoid way: there was no erosion of the bony walls of the tympanum. I did what I could in the region of the Eustachian tube, and after I had got into the carotid canal I judged that discretion was the better part of valour, and did not proceed further there. The points common to this case and Mr. Scott's are: the rapid growth, the destruction of the floor of the meatus, and the absence of any glandular involvement. The most important surgical question is, what should be done in the neck if one is satisfied that the primary growth has been cleared away? In my case I could not satisfy myself on that point. The patient was a stout and flabby woman, past middle age. What I believe is right in these cases is a procedure which I have employed a certain number of times, and I am interested to see that Mr. Wilfred Trotter advocates it. If there are no palpable glands, do an ordinary clearance operation but leave the sternomastoid. If there are such glands, clear everything, including the whole length of the sternomastoid muscles, the floors of both triangles, if necessary the submaxillary and parts of the parotid salivary glands, the internal jugular vein, and the whole of the overlying lymphatic structures, in a single sheet, and if possible without seeing the surface of any lymphatic gland. This method gives very good exposure, and there is astonishingly little loss of function. In one case so treated, you would not know that he had lost anything in the way of function in the neck and shoulder. You can be fairly certain, not quite, that there will be no glandular recurrence in the neck. The attempt to follow along the Eustachian tube appals me. Has anyone been bold enough to make it, save with the cautious use of a curette? I should like to make the attempt, but lack the courage.

Mr. W. M. MOLLISON: One explanation of the absence of pain while the disease was active may be, that pain in these cases is due solely to the pressure exerted by the growth when in a closed cavity.

Mr. SYDNEY SCOTT (in reply): I do not remember any case of malignant disease of the ear free from recurrence for more than four years, except the case of rodent ulcer, but my experience is limited to twenty-one altogether. [The PRESIDENT: Were the twenty-one all carcinomatous?] They include two of rodent ulcer and four of sarcoma. An interesting point concerns the site and direction of recurrent growth, which is nearly always *in situ*, and locally very malignant. I find the disease invades the temporal fossa or involves the cervical region, and the logical procedure in the former case would be, to excise the temporal muscle, the temporo-mandibular joint, and part of the mandible, but I have not seen a case which justified this being done. I have of course, like Mr. West, removed the sternomastoid completely with the glands of both cervical triangles, but I still feel uncertain whether to practise this as a routine, for the disease does not always follow this downward direction.

Mr. C. E. WEST (answering Mr. Scott): I should leave the patient alone, watching the condition of the cervical glands very carefully. If they begin to be palpable, there will be still time to clear the neck.

(*May* 18, 1917.)

Another Case of Translabyrinthine Drainage for Streptococcal Lepto-meningitis, with Recovery.[1]

By SYDNEY SCOTT, M.S.

THE patient, G. G., is a governess, aged 24, who suffered from chronic suppurative otitis media, which had already been treated by the radical mastoid operation on each side, before she came under my observation in May, 1916, when the following history was obtained.

History: Intermittent bilateral otorrhœa and deafness began in childhood. Two years ago the left ear was treated by the radical mastoid operation by Mr. Muecke. Four months ago the right ear became painful and discharged, and, six weeks later, the radical mastoid operation was done on this side by Mr. Badgerow. The earache and discharge subsided, but the patient continued to complain of headache, dizziness, vomiting, and very loud tinnitus. She developed neurasthenia, with insomnia, and was profoundly depressed mentally

[1] The above is an almost parallel case to one recorded in the *Proc. Roy. Soc. Med.*, 1913, vii (Sect. Otol.), p. 4, and to another recorded in *ibid.*, ii, p. 11.

and showed determined suicidal tendencies. She had to be constantly watched, and, nine weeks afterwards, was examined by Dr. Hinds Howell, who was able to exclude disease of the central nervous system. A fortnight later Mr. Badgerow asked if I would take her under my care.

Condition before Exploration.—It was at this period (May 11, 1916) I noticed a slight internal squint of the right eye with right facial weakness, but in view of Dr. Howell's recent report this was not to be attributed to a ganglionic lesion. There was no spontaneous nystagmus or Rombergism, though the patient walked with apprehension. At this time the right mastoid-meatal cavity was occluded by scar tissue, *pressure on which caused vertical nystagmus downwards, with forced movements of the head and trunk backwards and to the right.* This "fistula symptom" was therefore unlike that usually met with in fistula of the external semicircular canal, or in the case of an unduly mobile stapes.

HEARING TESTS.

	Right		Left
Low tone range limit (Bezold)	55 d. v.	...	90 d. v.
High tone range limit (steel monochord) ...	36 cm.	(Air)	36 cm.
	16 ,,	(Bone)	16 ,,
Rinne	Negative		
Weber	Doubtful		
Schwabach	Confused by tinnitus		

Raised voice was heard and understood on both sides.

In face of the risks I agreed to explore the right ear, and on the morning of May 17, 1916, I re-opened the mastoid and tympanic cavities, removed the dense tissue and new bone which encroached upon the tympanum and meatus, and obtained a satisfactory exposure of the inner tympanic wall. No fistula either of the external semicircular canal or of the oval window was discernible, neither could any opening be discovered with the fine flexible probe. There was some erosion of the posterior part of the promontory of the cochlea, and of the Fallopian aqueduct, but the fossula rotunda was unrecognizable. I thereupon left the labyrinth alone. Next morning (May 18) the tinnitus was the same as ever, and *spontaneous nystagmus* now appeared for the first time, being directed to the left (there was no nystagmus the evening after the operation), so it seemed evident from the direction and time of onset of the nystagmus that the labyrinth was becoming in some way involved. The giddiness also increased, and any movement of the head provoked vomiting: the facial paresis was more marked. On May 19

I awaited events; the evening temperature rose to 102·2° F., and the pulse-rate to 110. Next morning (May 20) the temperature had fallen to 99° F., and the pulse-rate to 76, suggesting an improvement, but there was now definite cervical rigidity; Kernig's sign was obtainable, while the tendon reflexes were diminished though not abolished. Moreover, the cerebrospinal fluid, which was now withdrawn by lumbar puncture, was found to be already "cloudy" (a later report by Dr. Stansfeld stated that it contained an excess of polymorphonuclear cells and yielded a pure growth of streptococci on cultivation).

Labyrinth and Internal Auditory Meatus Opened. — After withdrawing 20 c.c. of cerebrospinal fluid the vestibule and cochlea were opened through the meatus, without disturbing the sutures of the mastoid incision, and with a narrow gouge a free communication was established between the vestibule and internal auditory meatus, through which cerebrospinal fluid escaped freely and continued so to do for several days.

The improvement which followed was rapid and continuous; in twenty-four hours the headache and cervical rigidity had disappeared; the cerebrospinal fluid, withdrawn daily by lumbar puncture, was under less pressure, only 7 c.c. being collected, and this was "less cloudy," and in seventy-two hours was clear. The temperature returned to 98° F. and pulse-rate to 80, no subsequent rise taking place. The patient left the hospital in three weeks, and at first experienced the usual phenomena following ablation of one labyrinth. The facial paresis and internal strabismus disappeared two or three weeks afterwards, but the tinnitus remained loud and persistent, quite unchecked by the operation for fully two or three months; subsequently it gradually ceased to trouble the patient, who in the meantime had become cheerful and well.

Note.—The purpose of recording this case is to emphasize the importance of *early diagnosis* of lepto-meningitis, and at the same time the value of *immediate drainage* of the meninges through the labyrinth, when the signs indicate that this has been the route of infection, just as was shown in the case which I first described in the *Archives of Otology*, 1908, xxxvii, p. 108.

DISCUSSION.

Dr. DAN MCKENZIE: As in this successful case, I also have been able to rescue several patients from the jaws of death by this and other means of draining the meningeal spaces. If the ease of the operation were better

known, I think more men would be disposed to tackle cases of meningitis. Meningitis is regarded practically as a death-warrant, and so, often, nothing is done beyond a radical mastoid operation. But cases like Mr. Scott's show how unjustifiable it is to allow the patient to die without making an attempt to drain. I heartily support Mr. Scott's procedure in instituting prompt drainage when the diagnosis has been made; and now it is possible to make the diagnosis sufficiently early to save the patient in many cases. To decline to drain in meningitis is no more justifiable than to decline to open the abdomen in peritonitis.

Mr. W. M. MOLLISON: I do not agree with Dr. McKenzie. I prefer to treat these cases of lepto-meningitis not by draining to the internal auditory meatus, but by making incisions into the dura mater. Those cases which I have saved have been saved in that way. I did not find opening the internal auditory meatus as easy as Dr. McKenzie suggests: possibly I did not use the right instruments. The facial nerve was always very worrying in such cases, and I found it very dangerous to establish really good drainage when I had got through. And I do not agree with Dr. McKenzie in opening through the meatus as soon as the diagnosis of meningitis is made, because so many of these early cases can be saved by continuous lumbar punctures: these—a trivial operation—during the first twenty-four hours may save the patient.

Mr. C. E. WEST: I agree, to some extent, with all the speakers. The operation of translabyrinthine drainage is a remarkably easy one. It takes a very brief time after finishing the radical mastoid operation, and in many of the cases efficient drainage can be readily established, which will last four or five days; in some cases it has lasted fourteen days. But only a small proportion of the meningitis cases I have treated in this way—or in any other way—recover. One has had stray recoveries by all methods except in that of incision of the dura mater. I have incised the dura mater at various points. If nothing be interposed between dura mater and brain, the brain becomes adherent, and if gauze is interposed, it becomes infiltrated with lymph, and drainage ceases. When once withdrawn, it is impossible to put the gauze back. There are adhesions all round between brain and dura mater; in fact I have carried out this procedure for the very purpose of establishing adhesions. I agree with Mr. Mollison that in early cases perhaps the simplest and most effective method is either continued thecal drainage or thecal lumbar puncture—both simple methods. After fairly extensive trial of every method of treating meningitis I have heard of, I am coming back to thecal drainage as being the most hopeful way of treating cases. This matter was discussed in our Section at the International Congress of Medicine, and the further I attempt to treat cases of meningitis, the more convinced I am that the real secret, as was stated on that occasion, is that the choroid plexus blocks out the antibody. There are practically no antibodies in the cerebrospinal fluid, and until you can get a high antibacterial content in the cerebrospinal fluid, either by introducing

it mechanically, or as an antiserum, or by altering the permeability of the plexus, the great bulk of the cases of meningitis will be lost.

Mr. SOMERVILLE HASTINGS: The only cases of meningitis in which I have experienced recovery have been those treated by repeated lumbar puncture. I should like to hear from Mr. Scott whether, with a normal labyrinth, he considers it justifiable to do translabyrinthine drainage. I should also like to know something of the method Mr. West employs for continuous thecal drainage, because I have never done it, and I would like to try it.

Mr. C. E. WEST (replying to Mr. Somerville Hastings): The method of continuous thecal drainage is exceedingly simple. It consists of the introduction of a suitable trochar cannula into the theca in the ordinary position of lumbar puncture. It gives the most extravagantly profuse drainage. The end of the trochar comes out in the centre of a large rubber sheet, and over that are placed sterile sponges or swabs. Periodically the sheet is opened out and the sponges replaced.

Mr. SYDNEY SCOTT (in reply): In answer to Mr. Somerville Hastings, as mentioned in the note at the end of my case, it seems to me that the chief indication for draining the basal meninges through the labyrinth occurs when the meningitis is preceded by labyrinthitis, as is so frequently the case in otitic meningitis. When the patient is *in extremis* when first seen, I think it the right procedure whether one knows the labyrinth to be affected or not.

Specimens and drawings illustrating the anatomy of the Fallopian canal were shown by Dr. DAN MCKENZIE.

A series of stereoscopic skiagrams of the temporal bone was exhibited by Dr. ROBERT KNOX.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

J. Y. W. MacALISTER

UNDER THE DIRECTION OF

THE EDITORIAL COMMITTEE

VOLUME THE TENTH

SESSION 1916-17

SECTION OF PATHOLOGY

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1917

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SECTION OF PATHOLOGY.

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LONDON:
JOHN BALE, SONS AND DANIELSSON, LTD.,
OXFORD HOUSE,
83-91, GREAT TITCHFIELD STREET, OXFORD STREET, W. 1.

Section of Pathology.

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(*December* 5, 1916.)

(Chairman—Dr. J. A. Murray.)

A Large Salivary Calculus from Khartoum, Sudan.

By J. B. Christopherson.

In an experience of fourteen years' medical work in the Sudan, I have come across three cases altogether, of salivary calculus impacted in Wharton's duct. On October 1, 1916, I came across the third calculus, which weighed 24 grm. ($\frac{4}{5}$ oz.), and was 4·3 cm. long, 3 cm. broad, and 8 cm. in circumference, and is that described in the present note.

A Greek, named P. K., carpenter, aged 42, came to me with a very evident enlargement of the submaxillary gland on the left side, which was hard and fixed, and not acutely painful. On looking inside the mouth there seemed to be a hardness running on the left side in an outward direction from the middle of the floor of mouth, approaching the jaw somewhere about the level of the last left molar. The ridge on the top was papillated, and it still appeared like a new growth commencing in the floor of the mouth without ulceration; but, noticing a good deal of salivation, and that the mucus was mixed with foul smelling pus, I found it coming from the neighbourhood of the opening of Wharton's duct, which was patulous, on the side of the frænum: the correct diagnosis then occurred to me, more especially as the man said he had had the swelling a long time (eighteen years). I put in a probe along Wharton's duct, and after pushing it for $1\frac{1}{2}$ in. I struck the calculus. This I removed next day through the mouth, though the operation was not so easy as might appear. In the first place, as it was by no means easy to ascertain its shape, and as it was a very large

stone (24 grm.) (*see* fig. 1), the incision had to be enlarged several times. The surface of the stone being rough, it was firmly embedded and fixed into the tissues around, and even when grasped it was by no means easy to deliver. Further the jaw could not be opened to its fullest extent, so there was a limited space to work in. However, it was seized at last by a pair of delicate, curved, long tooth-stump forceps, and pulled out. The large cavity remaining was swabbed out and temporarily filled in with gauze. Although saturated with septic, smelling, salivary secretion, I did nothing to the submaxillary gland; in a short time it assumed its normal size.

One of the marked features of the case, after the removal of the calculus, was the rapidity with which the man regained flesh, colour, and discarded his careworn expression. He had evidently for some years been suffering from a chronic septic intoxication from this highly septic submaxillary gland and duct.

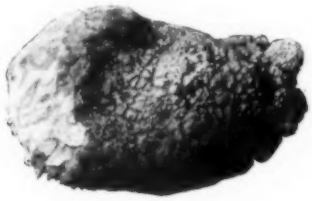


FIG. 1.

A photograph of the salivary calculus removed; natural size. The shallow groove marks the position of the ramus of the lower jaw.

The stone was of olive green colour (perhaps it had harboured the *Bacillus pyocyaneus*), and was mamillated on the surface. Its long axis was in the direction of Wharton's duct, and the small end nearest the orifice of the duct. It was, however, impacted $1\frac{1}{2}$ in. at least from the opening. There is a well marked groove in the stone, and this groove looked towards the ramus of the jaw; in fact the stone was grooved by the latter, so that it will be seen how much was above the level of the jaw, and how much below.

There was no means of saying that the man had a calculus, other than passing the probe down the duct and striking the stone, as it had in the course of so many (eighteen) years become surrounded by a thick envelope of connective tissue.

The history of the man is interesting. A native of Cyprus, aged 42, grey, dark, thin, sallow, septic looking, he was certain he had had the stone for eighteen years. He had consulted doctors in Cyprus about it eighteen years ago; altogether, four English and seven Greek doctors during his eleven years at Cyprus; and he had also been to the hospital at Nicosia for this swelling. During the last seven years spent in the

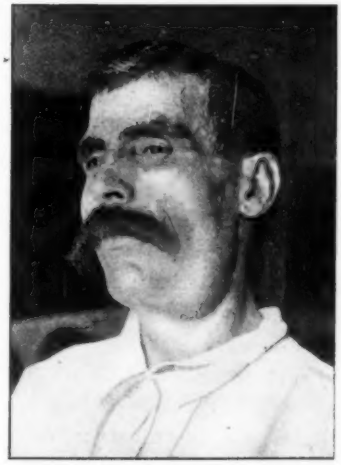


FIG. 2.

A photograph of the patient, showing the swelling in the side of the neck produced by the presence of the salivary calculus.

Sudan he had also consulted doctors, who had uniformly diagnosed it as a "gland in neck," and advised removal. He commenced consulting doctors five days after he had noticed the trouble, and had consulted them ever since. He says that the swelling had not increased in size lately; it had probably reached a maximum; and besides the swelling, he had complained of a profuse discharge of offensive and semi-purulent saliva in his mouth. His breathing was not affected, and swallowing was only interfered with when the mass became inflamed and big. Then it felt something like an egg being swallowed whole.

Examples of Ranula, Salivary Cysts, Salivary Calcu[illegible]n the Sudan.

The previous case occurred in a Greek; but, as one might expect in a dry tropical climate like the Sudan, where the excretory and the secretory glands work under a double disadvantage, the maximum amount of activity is required, and the minimum amount of fluid is obtained from the blood, since so much of the blood-fluid is dealt with by the sweat glands, causing concentration in the other secretions of the body. Calculi are hence common amongst the Natives, especially urinary calculi and salivary calculi; ranulæ and salivary cysts are also common.

In August, 1913, at Omdurman, I came across a patient who was a Sudanese Arab woman, aged 35, of the Dongolowi tribe. She had several ill-defined swellings in the left parotid region, and a large one in the left submaxillary region. There was also a cystic swelling the size of a large filbert in the substance of the tongue, on its left side. They were all said to have existed from infancy, and when I operated on them they seemed to be cystic spaces in the connective tissue, in fact cystic hygroma. The condition was, however, a rare form of cyst in connexion with the salivary gland.

Salivary calculi are, in the Sudan, oftener found in the parotid than in the submaxillary duct. Parotid calculi are usually small, often multiple, of varying sizes, and often associated with a cyst-like dilation of Stenson's duct. Those found in connexion with Wharton's duct have been, in my experience, single, and larger than the parotid.

Ranula due to the obstruction of the orifice of Wharton's duct is a common affection, filling up the oral cavity and displacing the tongue, and forcing down the sublingual tissues of the neck, giving the appearance of "double chin."

The appearance inside the mouth is very characteristic. The pink S-shaped curves of the lingual glands, run outwards and backwards from the middle front of the floor of the mouth, lying on the soft, blue bed of the cyst, which shrinks after the thick colourless or brownish thick glutinous secretion has escaped (2 oz. or more sometimes), the thin bluish membrane afterwards regaining the characters of normal mucosa.

In the case of ranula it is frequently found that periodically the orifice seems to become patent and the contents of the cyst discharged, and that the lower front teeth may be displaced by the pressure of the cyst.

(*December* 5, 1916.)

Note on an Amœboid Body found in the Urine.

By Dr. A. R. Friel, Captain S.A.A.M.C.

The specimens, which consist of the centrifuged deposit of urines, contain cells which were described in the *British Medical Journal* of April 8, 1916, p. 526, and were provisionally named "Amœba Urinæ Granulata." This body is present in the urine of great numbers of soldiers, in patients and in men apparently quite well. It is possible that it is of clinical as well as of scientific interest. The cell as present in the urine may be described as a *spherical* body, varying in size from that of an ordinary leucocyte, as seen in urine, to a body five times that in diameter, the average being 25 μ. It is, when viewed by daylight, of a *greenish* tinge, and much more refractile than a leucocyte. It can be readily picked out with a $\frac{2}{3}$ in. objective. It must not be confounded with an epithelial cell. Frequently there is a distinctly laminated wall. In the cell there are often one or more small spheres which resemble clear vacuoles. The wall of the cell may rupture and the spheres may then escape. Often there is a clear semicircular protrusion resembling a pseudopodium, as if the rupturing were only partial. It was the appearing of this clear protrusion that made us call the body an amœba. It has been pointed out that it is probably not an amœba in the proper sense of the term. It resembles a vegetable cell, recalling the "protococcus" met with in stagnant water. The cell is coarsely granular, and this gives it, when viewed with the high power, a semi-opaque appearance. The granules are situated in or close to the surface. The cell stains diffusely with the basic aniline dyes. A similar cell has been seen in a kidney-section made from one patient in whom there were large numbers in the urine; and likewise in the contents of the stomach of another.

The possibility is suggested that cells of a body such as "protococcus" are taken into the alimentary canal, and that there they develop into zoospores which reach the blood-stream, and that these, arrested by the kidney, develop into the bodies found in the urine.

(*December* 5, 1916.)

Pseudotuberculoma silicoticum of the Lip.

By S. G. Shattock.

Pseudotuberculoma silicoticum.

Summarium.

Hoc nomen idoneum esse videtur ad designandam laesionem quamdam rarissimam quae laesionem tuberculosis propriam insigniter simulat, cujus origo tamen mechanica solum fuit.

Tumor qui labio inferiori excisus est, in labio medio sub membranâ mucosâ jacebat.

Construitur omnino ex endothelii cellularum nodulis, cellulis ingentibus praeditis, ut in laesione tuberculosis propriâ. In plurimis e cellulis ingentibus includuntur silicis particulae quae, quum sectio polarizatur, per totam scintillant.

In glandulâ lymphaticâ submentali quae cum laesione labii excisa erat, scrutatio microscopica silicis particulas quoque demonstrat, quae laesione labii in glandulam translatae sunt.

Reactio tuberculosis propriâ (von Pirquet) abfuit.

Absunt quoque et bacilli tuberculosis et alia bacteria a sectionibus microscopicis secundum artem praeparatis.

Particulas ex silice constare ex hoc probatum est, quod quum tumoris fragmentum in acido nitrico calefacto solvitur, omnes perstant.

Aeger, homo omnino sanus et athleticus, ante multos annos praecipitatus erat in viam, facie suâ laceratâ et labio inferiore intus ab ore.

Non dubitari potest quin silex tunc in labii vulnus introductus fuerit.

In a previous communication[1] I have advocated the retention of the term "pseudotuberculosis" to denominate nodular lesions of a non-infective ætiology, which simulate those of tuberculosis. The multiple lesions of the lung occurring in different forms of pneumonoconiosis are the best known examples of such. In the testicle tuberculoid lesions,

[1] *Proc. Roy. Soc. Med.*, 1914, vii (Sect. Path.), p. 147.

again, may result from hyaline thickening of the walls of groups of the seminiferous tubuli in the senescent organ, accompanied, it may be, with hyaline change in the intervening stroma. Here the result is possibly due to the toxic action of effete products resulting from the disintegration of the tubular epithelium.

In the singular, and so far as I know unique, case recorded in the present communication, the lesion which clinically simulated a small neoplasm is histologically so remarkably like a tubercular lesion that it may be appropriately named a pseudotuberculoma. It was removed from the substance of the lower lip, by Mr. C. R. Nitch, of a patient in St. Thomas's Home. The history is that the patient, a healthy, athletic man, had been thrown from a bicycle whilst running down hill, on a country road, at high speed, in 1905, when he received injuries to

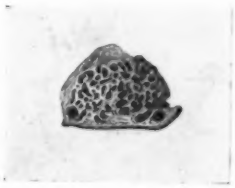


FIG. 1.

The silicotic pseudotuberculoma described, as viewed in mesial section, showing its actual size. On the lower side is the intact mucosa of the lip.

Pseudotuberculoma labii silicoticum. Sectio sagittalis magnitudinem tumoris ipsam monstrans. Membranâ mucosâ intactâ tegitur tumor.

the face including the inside of the lower lip; he distinctly remembers spitting out blood and sand after he picked himself up. He used a mouth-wash, and the healing of the injury to the lip took place in about a fortnight. For about a year before the operation, which was carried out on May 20, 1916, the patient had almost unconsciously acquired a habit of feeling over the inner side of the lower lip with his tongue, and early in 1916 he became aware of a lump in this position, which he associated in his mind with the receipt of the injury. On the inner aspect of the lip, just above the frænum, and slightly to the left of the mid-line, there was a firm rounded tumour over which the skin was freely movable. As felt through the skin the impression was like that given by a preputial chancre. The clinical diagnosis was uncertain, and as an alternative to some form of neoplasm Mr. Nitch sagaciously

suspected that the lump might be an indurative lesion set up by the inclusion of some foreign material. It was excised from the buccal aspect, with the overlying mucous membrane and the two submental glands which were slightly enlarged. Healing was uneventful, and was not followed by recurrence.

When divided after its removal, the formation was found to be circumscribed, with a somewhat sinuous outline; and it presented a granular aspect of unusual character. Its actual size in section is shown in the accompanying figure. It lay in the substance of the lip, close beneath the mucosa, which is uninvolved, and which, in the plane of section, presents no trace of injury, although the membrane must, of course, have been somewhere perforated at the time of the accident.

FIG. 2.

A microscopic section of the silicotic pseudotuberculoma described. It is constructed of an aggregate of typical giant-celled systems, and giant cells. In many of the giant cells there are included particles of silica, and there are smaller particles in some of the endothelial cells, and elsewhere. From an unstained section mounted in Farrant's medium. ($\frac{1}{2}$ obj.)

Pseudotuberculomatis labii sectio microscopica. Construitur tumor ex endothelii cellularum nodulis, cellulis ingentibus praeditis, ut in laesione tuberculosis propriâ. In omnibus cellulis ingentibus includuntur silicis particulae. Sectio in glycerino examinata paulum magnificatur.

MICROSCOPICAL ANATOMY.

In its microscopic structure the formation is particularly uniform, and consists of a close congeries of large multinuclear giant cells, of giant-celled systems, and of aggregates of systems, such as construct the miliary

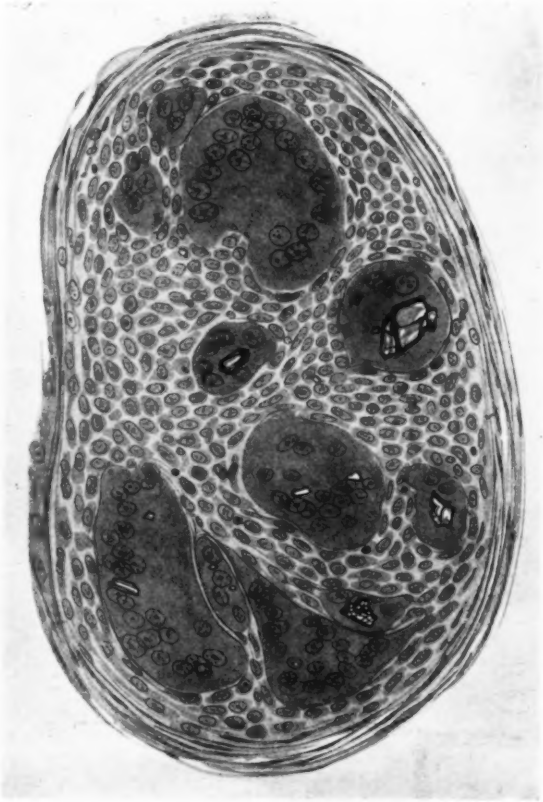


FIG. 3.

A group of multinucleated giant cells distributed in a circumscribed aggregate of proliferated connective tissue cells, the whole being surrounded with a narrow zone of fibrous tissue, as in a miliary tubercle. Many of the giant cells enclose particles of silica.

Pseudotuberculoma silicoticum. Monstratur e cellularum nodulis unus per quem sparguntur cellulae ingentes quae silicis particulas incluserunt.

tubercles of a tubercular focus. The accompanying figures will exhibit at once the structure indicated. Some of the giant cells are of striking size, and are surrounded with a narrow zone of fibre like the more typical

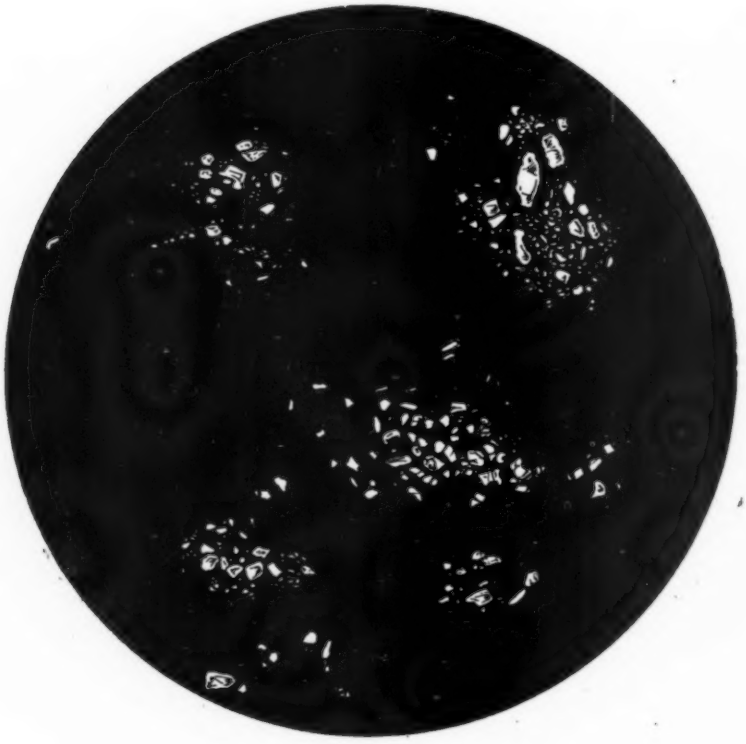


FIG. 4.

A section after treatment with strong nitric acid, which, being unheated, did not dissolve the tissue; as seen under the polariscope. The particles of silica, which are quite unaffected by the acid, show up brilliantly on the black background of the tissue in which they lie. (⅔ obj.)

Pseudotuberculomatis silicotici sectio microscopica ut apparet quum polarizatur. Silicis particulae in tumoris cellulis inclusae per sectionem totam scintillant.

giant-celled systems, which are composed after the usual manner, of a central giant cell and a zone of connective or endothelial elements. Regularly distributed throughout the new formation there are large

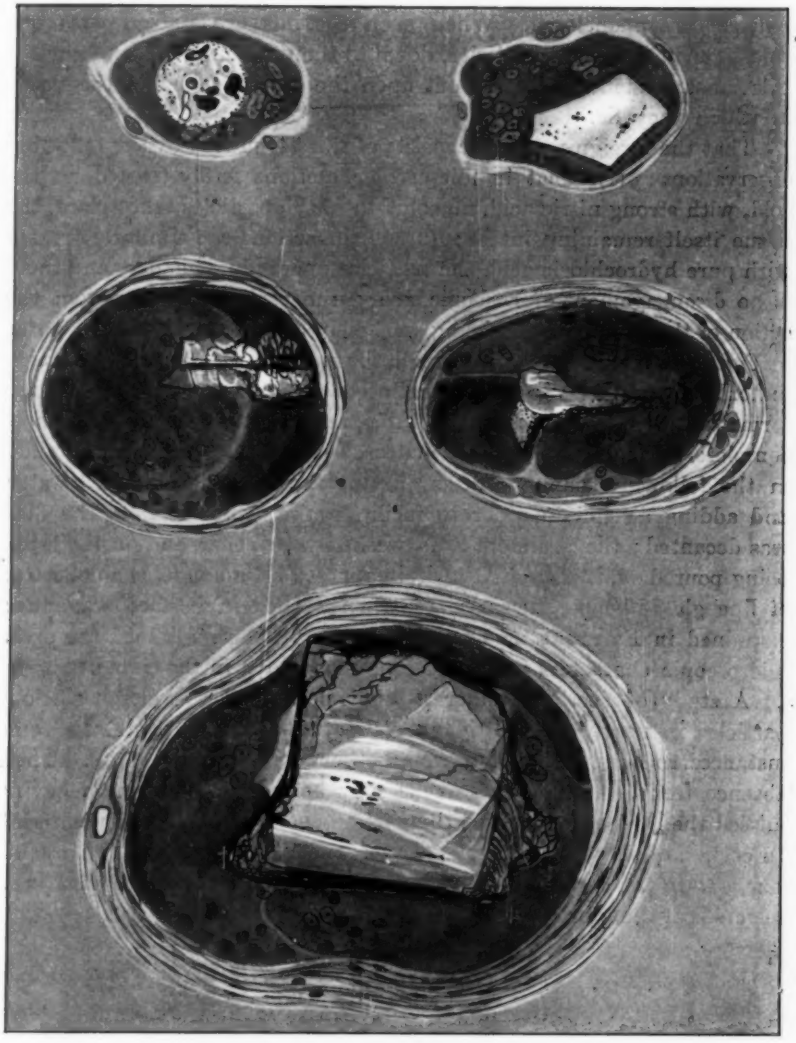


FIG. 5.

A selection of giant cells with included particles of silica of varying dimensions.

Cellulae quaedam ingentes e sectione microscopicâ selectae. In cellulâ quâque includitur silicis particula.

numbers of transparent, angular, colourless particles. Many of the giant cells include one or more such, and other particles lie in the surrounding cells, or are unenclosed. When such sections are studied with the polariscope the several particles, down to the most minute, are found to polarize, and stand brilliantly out upon the black ground of the tissue in which they lie.

That the foreign material consists of silica is proved by the following observations: (1) That in microscopic sections freely treated, in the cold, with strong nitric acid, no solution of the particles takes place, the tissue itself remaining intact; (2) when sections are similarly treated with pure hydrochloric acid, the particles undergo no change, and there is no disengagement of carbonic acid; the section shrivels from the action of the acid, but after being washed in distilled water and mounted in Farrant's medium, it displays all the original structure, the foreign material remaining absolutely unaltered, and (3), when portion of the formation was boiled in a test tube, in strong nitric acid, so as to completely dissolve the organic matter, I was able to recover the silica in the following manner: after diluting the acid with distilled water, and adding alcohol in order to facilitate the sedimentation, the fluid was decanted; the sediment was washed with ether, and on the latter being poured off, it presented itself, in appreciable amount, as a collection of fine glass-like particles: these, when washed in distilled water and examined in Farrant's medium, were found to have retained the exact microscopic characters which they present whilst in the tissue.

A study of sections under a high power ($\frac{1}{12}$) discloses silicious particles in nearly all the giant cells, the particles being, in some instances, so minute that they would otherwise escape detection. Their absence from some of these cells may be due to the section having missed the centre, or removed the portion enclosing the foreign material. In certain of these gigantic phagocytes, the particles are of remarkable size, and in the accompanying figure are shown some which have been selected for illustration.

Pathogenesis.

The production of this curious lesion must be ascribed to the forcible intrusion of silica (as sand) into the substance of the lip, from the injury recorded in the history. The formation is a non-infective granuloma, of a tuberculoid kind, and has the same non-infective ætiology as the pseudotubercles or dust-knots in the lungs of miners. Although of

some standing, it is a fact not to be passed by that no necrosis or caseation has anywhere taken place in the tissue, a thing, of course, congruous with its non-tubercular nature: the nuclei of the giant cells, again, show little of the circular or semi-circular arrangement so common in the giant cells of a tubercular focus. Nevertheless, so

FIG. 6.

A polarized microscopic section of one of the submental lymphatic glands from the same case, showing a group of minute particles of silica upon the dark background of the tissue, which have reached the gland from the lesion of the lip. ($\frac{1}{6}$ obj.)

Glandulae lymphaticae submentalis sectio microscopica ut apparet quum polarizatur. In glandulae telâ scintillant particulae quae ab laesione labii per canales lymphaticas in glandulam translatae sunt.

remarkably is the whole like a local tubercular disease that the latter has to be rigidly excluded; for the lesion might, conceivably, be of a mixed kind, the result of a local introduction of tubercle bacilli together with the foreign inorganic material.

Mr. Nitch was good enough, at my suggestion, to have von Pirquet's test carried out after the nature of the lesion had been discovered. This was done three weeks from the date of operation; the result

was quite negative. With the same object, I stained many of the sections by the Ziehl-Neelsen, Gram, and other methods (carbol thionine) without discovering a single micro-organism of any kind whatever.

In addition to the swelling in the lip, there were excised the two submental lymphatic glands, which were slightly enlarged. The minute examination of one of these fully confirms the view advanced in regard to the pathogenesis of the labial lesion. For in the polarized microscopic sections, the presence of a few small groups of silica particles is made obvious. The particles are of minute size as compared with most of those in the lip, and they appear to have been carried passively by the lymph current to the gland, later on in the history of the case, for there are none of the giant cells or giant-celled systems in the gland like those in the primary lesion.

As I have elsewhere remarked, foreign particles ingested by cells may, conceivably, be discharged into the connective tissue at the spot in which they were once intussuscepted; and they might thus find their way into lymph channels, along which they might be carried to the neighbouring lymphatic glands.

Glass Poisoning.

The only other form of traumatic lesion which suggests itself for consideration in the present connexion is that which might result from the administration of powdered glass: a common practice in India for homicidal and suicidal purposes. I may here cite some remarks made in connexion with the traumatic causation of appendicitis in the preceding volume,[1] as bearing upon this matter:—

"Certain results following the ingestion of powdered glass are known from this material having been used for criminal purposes. Unless, however, it is coarsely pounded, nothing harmful ensues under such circumstances; the one case cited in Taylor's 'Medical Jurisprudence'[2] being that of an infant who was killed by the administration of a quantity of roughly powdered glass, a considerable amount of which was found after death in the stomach, which was coated with a layer of tenacious mucus streaked with blood.

[1] S. G. Shattock, *Proc. Roy. Soc. Med.*, 1916, ix (Sect. Path.), p. 26.

[2] Fifth edition, by Dr. F. J. Smith, 1905, ii, p. 847.

"Dr. A. Mavrogordato tells me that he has recently given finely-powdered glass, &c., in food to guinea-pigs without producing any result; the administration extended over a period of one month. Three animals were used: one was given coal-dust, one flue-dust (the dust from the chimneys connected with engines driving mining plant), and another powdered flint; in the case of the last, powdered glass was several times substituted: no lesions were discovered in the alimentary canal, or in the mesenteric glands. These results confirm the findings of Le Sauvage[1] who himself swallowed a considerable number of glass particles without experiencing any inconvenience; 2½ dr. were given to a cat without subsequent injury; and 6 oz. and 7 oz. in eight days to a dog, with similar negative results. In India the administration of powdered glass for criminal purposes, homicidal and suicidal, is of well-known occurrence. If it is attended with a fatal result, this must be rare, for a reference to the well-known work[2] by Dr. Norman Chevers will show that none such is detailed; and in several instances in which glass has been crushed with the teeth and eaten by would-be suicides, mountebanks, and drunkards, either no symptoms have followed, or only temporary inconvenience. In the more recent publication of Dr. I. B. Lyon,[3] one fatal case, in which the cause of death was authenticated, is detailed, viz., that of a man who was attacked with symptoms of irritant poisoning and died in forty-eight hours; the gastric mucosa was reddened, and a quantity of powdered glass was found in the contents of the stomach; no irritant substance other than powdered glass could be found in the viscera."

It will appear from the foregoing how scanty are the pathological data in regard to this subject; and such few observations as have been made, are confined to the acute and readily investigated cases. That chronic forms of glass poisoning occur, is a widespread belief in India; cases, i.e., where the irritant induces a chronic intestinal disease which may terminate fatally. The pseudotubercular lesion described in the lip, and those found in the lungs of miners, however, can hardly be anticipated in the intestine, where abrasions could not but become infected, and where particles of glass if superficially introduced would be afterwards extruded by suppuration. It may be doubted, indeed, whether there is any form of intestinal silicosis comparable with that of the lung.

[1] "Inaugural Dissertation," Par., 1820.

[2] "Medical Jurisprudence of India," 3rd ed., 1870, p. 287.

[3] "Medical Jurisprudence for India," 1889, p. 229.

Through the kindness of Sir Arbuthnot Lane, I have recently had the opportunity of examining a portion of the ileum excised by him, in a case where the general evidence makes it probable that the disease was due to glass poisoning, and which I may recount as a conclusion to the present communication. The piece of ileum, 20 cm. (8 in.) in length, was excised, with a completely successful result, from a patient who was dying from some obscure form of intestinal disease accompanied with obstruction. After the abdomen was opened the site of obstruction was removed, together with a length of thickened bowel above, an ileosigmoidostomy being then carried out: an œsophageal tube was passed *per rectum* through the anastomosis, the free end of the tube resting in the ileum 6 in. beyond the new junction. The patient, a British officer, was invalided home from India, and furnished the following history. He was seized with pain, accompanied with diarrhœa and vomiting after a meal (in March, 1916), and from that date he suffered from recurrent attacks of abdominal pain, to which obstruction was later superadded; the general condition becoming eventually so grave that an abdominal exploration was carried out, and the affected portion of the intestine excised. The evidence of poisoning was the admission of an Indian servant that after receiving his discharge, he had concealed powdered glass in a meringue, which was unsuspectingly taken by the patient, who until this, had been a perfectly healthy man. The piece of bowel removed (in June, 1916) was 20 cm. (8 in.) in length, and before being slit up, it was obviously thickened, and abnormally stiff, but without being generally contracted; for a short distance the proximal end was dilated as a secondary result of obstruction beyond. When divided, the following conditions presented themselves: For the distal 14 cm. (5½ in.), the mucosa is swollen, the valvulæ conniventes being unnaturally voluminous, and about the distal end the membrane is ulcerated, the lumen being here strictured; one of the valvulæ conniventes in the same area is undermined so as to form a bridle. There is a second, minor constriction of the bowel at the other end in connexion with a second area of ulceration; above this the gut is dilated, the muscularis having undergone a secondary, compensatory hypertrophy. Excluding the dilatation, the submucosa is slightly thickened as compared with that of portion of the intestine from the same region of a healthy, well-developed adult, aged 35, who committed suicide. The muscularis is notably hypertrophied, the general condition recalling in some degree that seen in a leather bottle stomach. The anatomical condition is, in short, one of chronic enteritis accompanied

with some local ulceration and ensuing stricture (fig. 6). The microscopic sections of the intestine, which were cut transversely, and made so as to include the ulcer at the distal end, show that the latter is covered with a well-defined layer of normal granulation tissue, the mucosa itself having been quite destroyed. In a longitudinal section of the thickened wall made at some distance from the ulcer, I found a small circular granulating hole in the submucous coat, probably part of a track communicating elsewhere with the lumen. The submucosa (and the corresponding middle tissue of the valvulæ) presents the usual picture of small-celled infiltration, the elements including a conspicuous number of eosinophile polymorphs. There is nothing whatever suggestive of tuberculosis. No particles of glass were encountered in any of the sections, and there was an absence of bacteria, Gram-positive or other, both in the granulation-tissue of the ulcer and in the deeper wall of the gut. In the divided edge of the mesentery, there was exposed a lymphatic gland, the size of a pea. Microscopic sections of this showed it to be quite normal in structure: it contained no glass, and no bacteria.

In commenting upon this case, we cannot go further than to say that there is nothing incompatible with the lesion having been set up by ingested glass. The disease is not tubercular, and the history is sufficient to exclude its being syphilitic. The difficulty of explaining the kind of lesion, and its position, in the ileum, on any other hypothesis (than that of glass poisoning) must likewise be taken into account, and as before observed, a chronic disease set up by injury from powdered glass might be expected to take such a form; it would be essentially an infective process following upon local abrasion, terminating, it may be, in some degree of ulceration, and eventually in stricture.

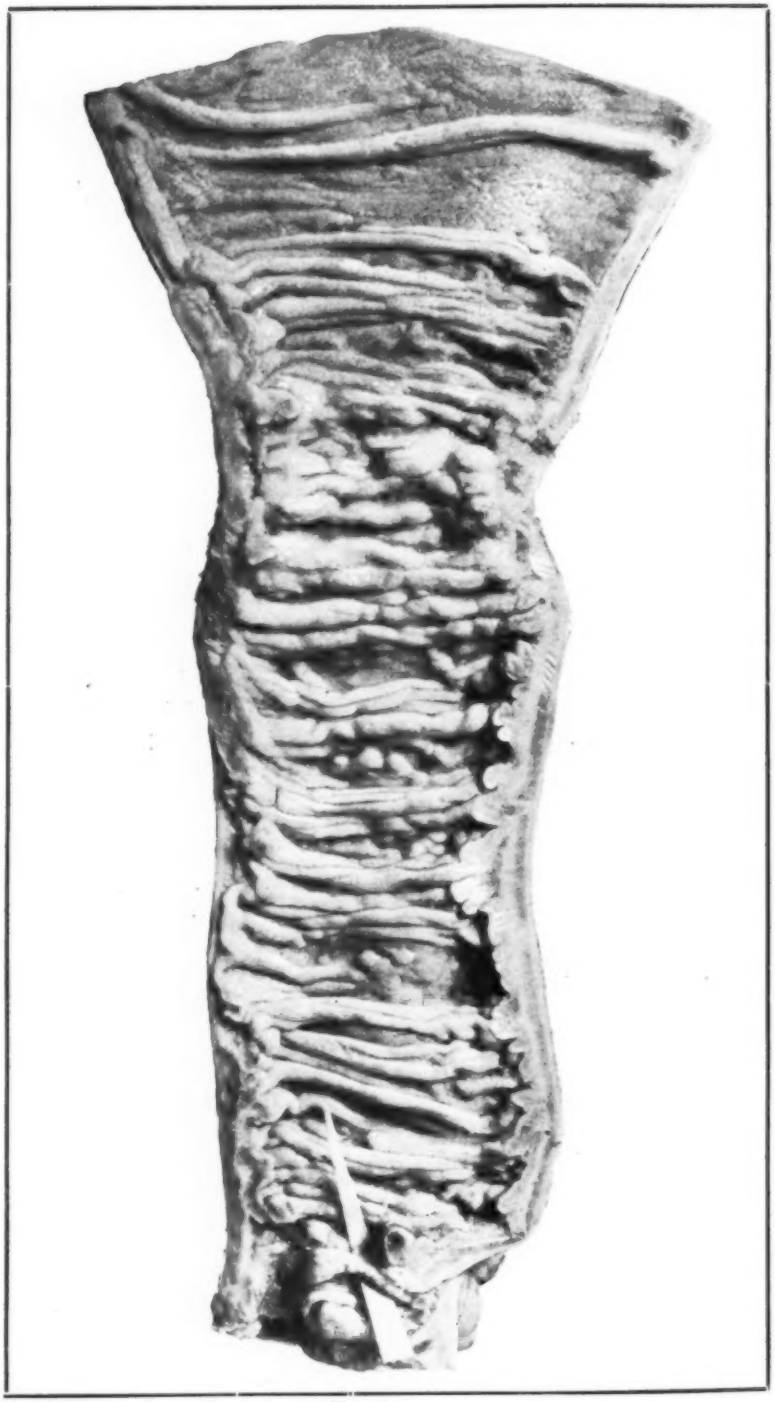


Fig. 7.

DESCRIPTION OF FIG. 7.

A portion from the lower half of the ileum, excised in the presumptive case of glass poisoning referred to in the text. For nearly its whole length the mucosa is tumid, the valvulæ conniventes being unnaturally voluminous; at the distal end (the lowest in the figure) the mucous membrane is ulcerated, and one of the valvulæ conniventes has been undermined so as to form a bridle, beneath which a triangular slip of paper has been passed; at this spot the bowel is strictured. The muscular coat is notably hypertrophied. There is a second, minor constriction towards the upper end; above this the bowel is dilated. Natural size.

Intestini ilei portio excisa et laesionibus affecta quae vitro contuso glutiendo forsitan causatae sunt, videlicet: membranae mucosae tumiditas; membranae illius apud extremitatem intestini inferiorem ulcus; unius valvulae conniventis disjunctio partialis; luminis illic constrictio; tunicae muscularis crassitudinis augmen.

(*December* 5, 1916.)

Grafting Experiments made with Normal Mouse Tissues treated with Cell-free Extract of Mouse Cancer, or admixed with the Tumour Pulp, &c.

By S. G. Shattock and L. S. Dudgeon.

Summarium.

Factae sunt hae observationes ut determinaretur num muris carcinomatis aut (1) extractum aut (2) cellulae tumoris ipsae incitarent telas foetales ad neoplasmata generanda.

(1) E carcinomate praeparatum est extractum tumore cum vitro contuso in liquore Ringeri agitato. Muris foetus minute persectus in hoc extracto maceratus est per unam horam. Liquore deinde defuso foetus pulpa sub murium cutem inserta est. E telis foetalibus sic insertis neoplasma nullum ortum est.

(2) In altero experimento muris carcinoma cum foetu persectum sub cutem insertum est. Quindecies subcultus est tumor, tumoribus interdum cum murium foetibus persectis. Telarum foetalium nullae in mediis tumoribus crescentibus perstiterunt nisi cartilaginis quaedam fragmenta ; nullae, autem, notae metamorphosis sarcomatosae in his apparebant.

Augmen, quidem, cartilaginis foetalis constanter evenit postquam sub cutem inserta est : hoc observatum est in experimento quoque quod primum citatum est.

The investigations detailed in the present communication, although the results only add to the body of what may be called negative science, are perhaps worth recording, if merely to save their needless repetition in the hands of others. They furnish negative answers to certain questions which have presented themselves as possibilities demanding the test of experiment. The particular mouse tumour used was that known as No. 63, at the Imperial Cancer Research, for the supply of some of the stock of which we desire to express our obligations to the Director, Dr. E. F. Bashford, and to his successor Dr. J. A. Murray. One defect from which the experiments suffer is the low malignancy of this particular tumour. For whilst this does not detract from the value of the results arrived at by the Imperial Cancer Research, in regard to

studies in cellular immunity, it undoubtedly detracts from the experiments recorded in the following communication.

The term "mouse cancer" must be taken to comprise, as indeed it really does, any form of malignant tumour arising in mice, of whatever histological class or grade of malignancy. From certain histological appearances presented by some of the grafted tumours in the present series, we do not ourselves feel sure that this particular tumour, No. 63, is a carcinoma—i.e., a malignant growth of epithelial origin. The fact that the original neoplasm arose in the mouse's mamma, is, of course, equally compatible with the histological view that it may be a periangial sarcoma or malignant perithelioma.

The object underlying the experiments was to ascertain whether cancer cells possessed any biological properties which would incite normal cells to participate in their purposeless or anarchical growth. For this end a cell-free extract of mouse cancer was prepared by shaking the tumour-pulp (made with the mincer) in Ringer's fluid, to which beads and fragments of glass had been added, so as to destroy the cells: the material was then lightly centrifuged in order to bring down the coarser débris. The cell-free extract, having been pipetted off, was used as a medium in which to soak the normal tissues, which were then grafted subcutaneously. That the extract was incapable *per se* of producing a tumour was shown by the fact that the centrifugalized sediment of the tumours so shaken proved to be sterile when inserted subcutaneously: eight mice were so treated, and all with a negative result, three being killed after twenty-six days; and five, after twenty-five days. The normal tissues used were (1) mouse testicle: and (2) in order to comprise the whole group with their endocrinal interactivities, mouse embryo. As a further, and perhaps better, extension of the second experiment, the tumour itself was mixed with mouse embryo, the two being placed in the mincer and cut up together. Fœtal tissue of every kind was thus implanted amidst the growing elements of the tumour; our object being, as before stated, to see whether any would be incited to grow after the manner witnessed, e.g., in the well-known malignant enbryoma or "mixed tumour" of the human testicle. As will appear, the only positive result attained was limited to the persistence and some growth of the cartilage admixed with the tumour. Although the tumour with which the experiment was commenced, was subcultured fifteen times, the investigation extending over a period of nearly twelve months, and although the subcultures were "rejuvenated" on three occasions with

mouse embryo, nothing of higher interest ensued; the cartilage had no sarcomatous character impressed upon it; and the very last of the fifteen subcultures presented otherwise nothing either macroscopically or microscopically to distinguish it from an ordinary growth of this particular neoplasm. No visceral metastasis took place on any occasion.

The result noticed, indeed, must itself only be regarded as an independent and transitory growth of the grafted cartilage, none of the other embryonic tissues having survived or "struck." It is of the same kind as the transitory growth of cartilage which we found to occur when minced mouse embryo, after treatment with cell-free cancer extract, was inserted beneath the skin. In a previous communication[1] we have fully described the enlargement of the epiphyseal cartilage at the ends of fœtal long bones isolated from the rabbit embryo, and inserted, entire, beneath the skin of the mother, or of the unrelated adult rabbit: this, again, is an illustration of the vitality and power of growth possessed by cartilage under transplantation, although the limitation of the growth marks it off from what is witnessed in a proper chondroma. Finally, in two experiments the cell-free extract was used as a subcutaneous injection immediately before the introduction of the untreated pulp of a similar tumour, at the same spot. And in another experiment the extract and the pulp were mixed and inserted simultaneously. The object of this experiment was to see whether the cell-free extract would act upon the connective tissue in such a way as to induce its chemiotactic invasion by the tumour under growth, and so render this particular neoplasm more invasive—i.e., more malignant.

The Subcutaneous Insertion of Minced Mouse Testicle which had been allowed to soak in a Cell-free Extract of Mouse Cancer.

As stated in the introductory remarks this experiment was designed to see whether the cells of the testicle would be incited to produce a growth as the result of the action of the cancer extract.

April 8, 1915.

Two mouse tumours of fifteen days' growth were minced and shaken for three hours with glass, in Ringer's fluid; on April 9 the mixture was again shaken for four hours, and lightly centrifuged; the super-

[1] S. G. Shattock, C. G. Seligmann, and L. S. Dudgeon, *Proc. Roy. Soc. Med.*, 1910, iii (Sect. Path.), p. 127.

natant liquor was pipetted off, and in this were finely minced with curved scissors, the testicles of five mice, some of them sexually mature. The mixture was incubated at 37° C. for an hour, being stirred from time to time. Three mice were injected intraperitoneally with the fluid pipetted off from the mixture after incubation. Eight mice (mostly females) were injected subcutaneously with the solid residue. All the animals remained in perfect condition, no tumours appearing beneath the skin.

July 31.

The abdomen of a doe of the three which had received intraperitoneal injection had become enlarged (without pregnancy), and the animal was killed. The enlargement proved to be due to the presence of two symmetrical masses of fat which had grown about the uteri, probably in connexion with sexual senility.

August 2.

The other two animals which had received intraperitoneal injections were killed. Post-mortem: The peritoneum and all the viscera were normal. Four of those which had received subcutaneous injections were killed; none showed any local or visceral lesions.

September 13.

The remaining four of the animals injected subcutaneously were killed, the result being equally negative.

The Subcutaneous Insertion of Minced Mouse Embryo which had been allowed to soak in a Cell-free Extract of Mouse Cancer.

This experiment was designed for the purpose of ascertaining whether the embryonic tissues would be incited to produce a growth as the result of the action of the cancer extract.

June 4, 1915.

Three tumours of fourteen days' growth were shaken with glass in Ringer's fluid, &c., as before, the material being kept during the ensuing night in the ice-safe.

June 5.

A pregnant mouse was etherized and decapitated, the uteri removed, and two of the fœtuses were passed through the mincer, without placentæ or membranes. The pulp was then mixed with the cancer extract, and incubated at 37° C. for fifteen minutes. Three mice

received intraperitoneal injections of the fluid, which was pipetted off; and six mice were injected subcutaneously with the solid residue. The animals kept throughout in perfect health.

August 3.

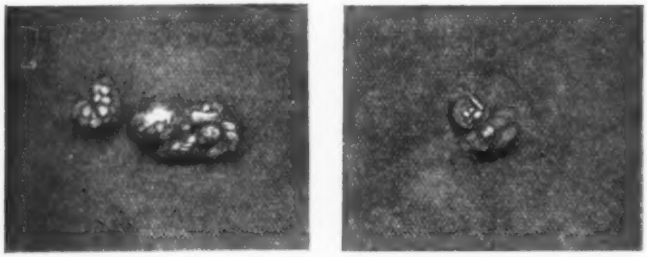
Three of the six injected subcutaneously were killed. On the site of injection there was a small amount of particulate débris.

October 22 (*Twenty Weeks*).

Two of the three injected intraperitoneally were killed. Post-mortem: The abdominal cavity and the viscera were quite normal. Two of the remaining three injected subcutaneously were killed; in each there was at the site of injection a small group of minute calcified nodules of cartilage. The viscera were all normal.

February 1, 1916 (*Thirty-four Weeks*).

The remaining animal injected intraperitoneally was killed. Post-mortem: It was in every way normal. The remaining mouse injected subcutaneously was killed. At the site of injection there was a small group of minute cartilaginous nodules, mostly calcified.



A FIG. 1. B

Two collections of minute processes of cartilage, mostly calcified, and representing the results of the subcutaneous insertion of mouse embryo, which, after having been minced, was mixed with cell-free extract of mouse cancer (Tumour No. 63, Imperial Cancer Research). Of the inserted tissues the cartilage alone has survived, and, to a certain extent, grown—a result not attributable, however, to the action of the cancer extract. In the case of the left-hand figure, A, the grafted material lay *in situ* for twenty weeks; in the other, B, for thirty-four weeks, before the animals were killed. (Enlarged twice.)

Monstrantur cartilaginis fragmenta plerumque calcificata quae muris foetalis percisi residua repraesentant, foetu sub cutem inserto postquam in extracto muris carcinomatis maceratus erat. E telis foetalibus neoplasma nullum ortum est. A, a mure interfecto post viginti hebdomadas. B, a mure interfecto post triginta quattuor hebdomadas. Figurae bis magnificantur.

June 18, 1915.

A repetition of this experiment was carried out by preparing the extract as before. With this the minced embryos were mixed on June 19, at 12.30 p.m., and incubated at 37° C. until 3.30, after which the mixture was stored in the ice safe until June 21, when the fluid was pipetted off, and the sediment used to inoculate six mice subcutaneously. The animals remained in perfect condition without displaying any local swelling.

August 3 (*Six Weeks*).

Three were killed; at the site of injection in one, there was a small group of calcified fragments; the other two showed no residua.

September 13.

The remaining three, which were killed to-day, proved equally negative as to tumour growth, and the different viscera were normal.

The Subcutaneous Injection of Mouse Cancer with which Minced Mouse Embryo had been mixed.

The design of this experiment was to see whether the embryonic tissues would be incited to grow amidst the carcinoma after the manner of the embryomatous or mixed tumour met with in the human testicle. The tumour was subcultured fifteen times in the course of the experiment, and was mixed with mouse embryo, altogether four times. The technique was rigidly aseptic, and in no case did bacterial infection take place.

July 10, 1915.

A tumour of thirteen days' growth was minced with a mouse embryo without the membranes or placenta; portions of the embryo (which was in bulk about equal to the tumour) being placed alternately with pieces of the tumour, in the barrel of the mincer. The fine reddish pulp which came through was further mixed, and used to inject eight mice subcutaneously.

July 23 (*Thirteen Days*).

In seven of the eight animals, tumours of varying sizes had grown, the largest being 2 cm. in chief diameter. A microscopic section of

one of these tumours, the animal being killed twenty-one days after the inoculation, showed that it was closely connected with the muscular wall of the abdomen through the medium of a zone of highly cellular

FIG. 2.

A microscopic section from the original tumour (No. 63, Imperial Cancer Research) which was mixed with minced mouse embryo, the animal being killed twenty-one days after its subcutaneous inoculation. In the growing periphery of the neoplasm there lie embedded portions of two fœtal bones; one, a shaft in longitudinal view; the other, portion of an epiphysis. The shaft is being destroyed by ingrowths of connective tissue, but no invasion of it by the cells of the neoplasm is in progress. The other fragment consists of hyaline cartilage, the cytoplasm and nuclei of the cells of which stain in a perfectly normal manner. No sarcomatous change has taken place in the cartilage, nor has it been invaded by the cells of the neoplasm. Immediately beyond the tumour and the embedded foetal fragments, is shown a cellular zone of connective tissue; and beyond this, portion of the muscular wall of the abdomen. ($\frac{2}{3}$ obj.)

Sectio microscopica muris carcinomatis, tumore antequam sub cutem insertus est, commixto cum muris foetū persecto. Interfectus est mus diebus post viginta quam inoculatus erat. In neoplasmatis margine includuntur duo fragmenta quae ossium partes foetalium repraesentant. Ossium horum cartilago neque cellulis neoplasmatis invaditur nec in sarcomatis telam transmutatur.

connective tissue, but without evincing any indications of invading it. Centrally the tumour was necrosed and cystic, the spaces being filled with a homogeneous uncoloured substance, a coagulum of fluid brought

about by the action of the formol solution in which the specimen had been fixed; beyond the necrotic substance there was a thin cortex of growing tumour-tissue. In the actual substance of the latter, on the deep aspect immediately adjacent to the proliferating connective tissue over the abdominal muscles, there are portions of two fœtal bones; in many spots the cells of the neoplasm are in immediate contact with the cartilage, but no invasion of the latter by the former has taken place. One of the fœtal bones is obviously a shaft in longitudinal view, and this is undergoing destruction by the ingrowth of young connective tissue from points around, where it is not immediately invested with the cells of the tumour. The eroded shaft stains somewhat deeply with hæmatoxylin. The second, somewhat circular process close by the preceding, consists of hyaline cartilage, the cytoplasm and nuclei of the cells of which are in a perfectly normal condition, and give no indication of having undergone a sarcomatous change.

July 28 (*First Subculture*).

One of the mice was killed, and the tumour used to inoculate four normal animals.

August 17 (*Second Subculture*).

From one of the preceding mice the tumour was used to inoculate three further animals. Microscopic sections of another tumour from the mice inoculated on July 28, and killed on August 11 (fourteen days) showed a thin rind of normal growth, with here and there a fibrous septum passing for a short distance from the exterior; the rest was necrotic and hæmorrhagic. In this there were two small foci of cartilage; one of these consists of three secondary processes, in one of which the matrix stains deeply with hæmatoxylin, indicating that calcification was in progress. The nuclei of the cartilage cells are unstained, or represented by granules of deeply stained, broken down chromatin, showing that the fragments have undergone necrosis.

August 30 (*Third Subculture*).

November 2 (*Fourth Subculture*).

November 24 (*Fifth Subculture*).

December 16 (*Sixth Subculture*).

January 10, 1916 (*Seventh Subculture*).

In microscopic sections of one of the tumours from the inoculations carried out on December 16, no fœtal structures were seen, nor anything in which the neoplasm differed from the usual type.

January 26 (*Eighth Subculture*).

February 16 (*Ninth Subculture*).

Two tumours from the previous subculture were minced with one embryo (well under full term, 2 cm. in length), three mice being subcutaneously injected with the mixed pulp.

March 6 (*Tenth Subculture*).

A microscopic examination of one of the tumours raised from the inoculation of February 16, the animal being killed after sixteen days, showed a small oval process of cartilage with normal nuclei, lying in necrotic portion of the growth; to the perichondrium there are attached some pieces of striated muscle fibre; these, however, present no indications of growth.

The tumour from which the tenth subculture was made, was minced with mouse embryo of an early age, the embryos being 1 cm. in length.

March 23 (*Eleventh Subculture*).

April 12 (*Twelfth Subculture*).

April 28 (*Thirteenth Subculture*).

May 12 (*Fourteenth Subculture*).

June 1 (*Fifteenth and last Subculture*).

Two tumours of the fourteenth subculture were minced with two mouse embryos and one placenta, the embryos being 1 cm. in length. Four mice were injected subcutaneously.

June 21 (*Twenty Days*).

One of the four inoculated mice presented a full-sized growth and was killed; the tumour, which, on section, showed nothing unusual, was hardened in formol for microscopic examination. In the other three the tumours were of moderate size.

July 18 (*Seven Weeks*).

Another of the mice was killed, and the tumour, which was of full size, fixed in formol; its divided surface presented nothing unusual to the naked eye.

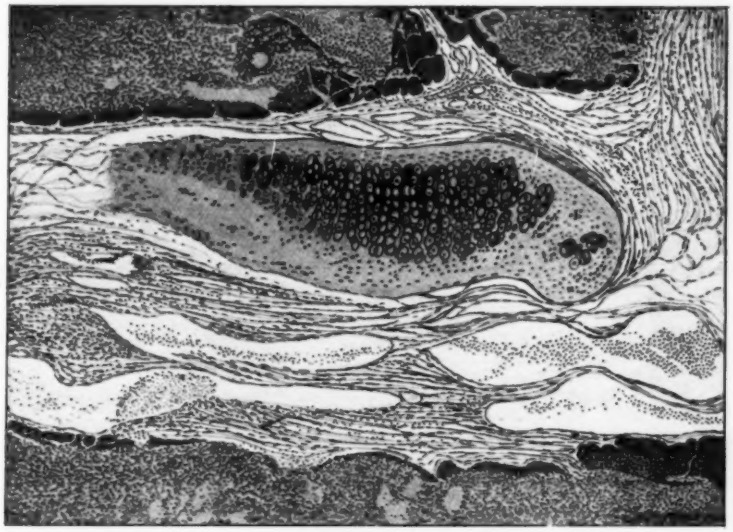


FIG. 3.

A microscopic section from the fifteenth subculture of mouse cancer (Tumour No. 63. Imperial Cancer Research) with which, during the course of propagation, minced mouse embryo had been mixed on four occasions. The last admixture of mouse embryo was made with the particular tumour of this fifteenth subculture. In the spot shown the grafted cartilage of the embryo has survived and grown to a small extent in one of the connective septa of the neoplasm, but without presenting any indication of having undergone a sarcomatous transformation. The tumour, which is of seven weeks' growth, is the seat of extensive necrosis and hæmorrhage. ($\frac{2}{3}$ obj.)

Sectio microscopica muris carcinomatis quod quindecies subcultum erat, tumoribus interdum commixtis cum murium foetibus persectis. In septo fibroso tumoris monstratur cartilaginis foetalis fragmentum, cujus e cellulis etsi multiplicantibus nulla laesio sarcomatosa orta est. Aliarum telarum foetūs nullae in medio tumore perstant. Interfectus est mus post septem hebdomadas.

Microscopic examination of the tumour of twenty days' growth: In its mid-substance, where necrotic, there is a small fragment of necrosed cartilage, without perichondrium; evidently a piece which has been

transferred, and has died. The neoplasm is the seat of extensive hæmorrhage; its healthy peripheral part exhibits a structure which admits of being regarded as peritheliomatous, the cells forming thick mantles about a central lumen.

Microscopic examination of the tumour of seven weeks' growth: The growth is extensively necrotic and hæmorrhagic; where normal it exhibits in places a distinctly peritheliomatous construction. In one of the fibrous septa, extending in from the surface, there is an elongated process of hyaline cartilage, furnished with perichondrium, the large size of the cells of which, and their serial arrangement, show that they are in process of proliferation. The matrix has in places undergone calcification as if preparatory to ossification. In line with this process, and apparently part of it, there is a second, lesser; and beyond the last, there are two still smaller, incomplete rings of deeply stained material, which are evidently cross sections of fœtal shafts; these are in process of replacement by connective tissue which fills and surrounds them; a certain number of osteoclasts lie against the inner sides of the vanishing shafts.

Remarks.

In the foregoing experiment the tumour was subcultured fifteen times, the observation extending over a period of about one year, and it was mixed with mouse embryo on four occasions—viz., July 10, 1915 (the original, or first culture); February 16, 1916 (ninth subculture); March 6, 1916 (tenth subculture); June 1, 1916 (fifteenth subculture). The admixture of the minced fœtus did not either accelerate or retard the growth of the tumour. Of the fœtal tissues the only one to survive and to exhibit a slight degree of increase, was the cartilage of the embryonic bones. But this died out under successive propagation; it underwent no sarcomatous transformation so as to bring about the growth of a mixed neoplasm, nor did it develop into processes like those met with in the embryomatous or mixed tumours of such well-known occurrence in the human testicle. Of the particular microscopic sections selected for illustration one is of the fifteenth and final subculture with which embryo was mixed. The animal was killed forty-nine days after subcutaneous inoculation. In one of the fibrous trabeculæ of the tumour there is shown a fragment of fœtal cartilage which has survived and grown to a limited extent. In the other section, taken from the original tumour, —i.e., the one to which the first admixture of embryo was made—the animal was allowed to live twenty-one days. The fragments of fœtal

cartilage here lie in immediate contact for a considerable way, with the normally growing cells of the neoplasm. Yet not only has this cartilage failed to undergo any sarcomatous transformation, but it is singular that the tumour cells have made no attempt to invade it. This is noteworthy, since if one were asked to adduce an instance in normal biology of the invasion and destruction of one tissue by another (which is essentially the phenomenon underlying malignant neoplasia), one would point to the normal disappearance of fœtal cartilage under the inroad of osteogenic connective tissue. But here nothing of an invasive process has been incited; there is nothing chemiotactic in the cartilaginous tissue, although, as pointed out at the beginning of the paper, the low grade of malignancy naturally exhibited by this particular mouse cancer must be borne in mind. The survival and transitory growth of the grafted cartilage when mixed with the tumour is, in fact, only what regularly takes place when fœtal cartilage is inserted beneath the skin of any animal of the same species.

The Injection of Mouse Cancer subcutaneously, twenty-four hours after the Injection at the same spot of an Extract of a Similar Tumour.

The object of this experiment was to ascertain whether any chemiotactic effect would be produced upon the connective and muscular tissues, which would render the tumour more invasive—i.e., more malignant.

May 20, 1915.

The extract prepared as usual, was injected subcutaneously in three mice.

May 21.

Freshly prepared tumour pulp was injected beneath the skin at the site of the previous introduction of extract. The animals remained in perfect condition, and oval tumours of fair size (averaging 1·5 cm. in longer diameter) had developed in all.

June 4 (*Fourteen Days*).

One was killed: the tumour was not adherent either to the skin or to the abdominal muscles.

June 15 (*Twenty-five Days*).

The remaining two were killed. In one the growth had retrograded and disappeared. The other presented a full-sized tumour which was not infiltrating any of the surrounding structures.

The Subcutaneous Injection of Mouse Cancer with which Extract had been Mixed.

The object of this experiment was the same as that of the preceding.

May 19, 1915.

An extract of two tumours of sixteen days' growth was prepared in the usual way.

May 20.

The fluid was pipetted off, and the pulp of a tumour of sixteen days' growth was mixed with it. Seven mice were subcutaneously injected with the mixture. They remained in perfect general condition.

June 3 (*Fourteen Days*).

Of the seven mice, five show well declared tumours, averaging the size of a haricot; the other two were negative.

June 10.

The tumours in the five mice had steadily progressed.

June 18.

Four of the mice bearing tumours were killed. In all, the tumour was adherent to the abdominal muscles over a small oval central area; but the peritoneum was not involved; the skin was unaffected. This degree of adhesion, however, is by no means uncommon under the common conditions of grafting with this particular tumour.

Of further questions with negative answers, we may throw together the following :—

CAN A DIMINUTION IN THE CAPACITY FOR GROWTH OF THE TUMOUR (No. 63)—I.E., AN ATTENUATION—BE BROUGHT ABOUT BY SUBCULTURING A SELECTED SUCCESSION OF BADLY GROWING TUMOURS; AND IF SO, WOULD THE GRAFTING OF SUCH A STRAIN AFFECT THE GROWTH OF A VIGOROUS NEOPLASM, WHEN USED CURATIVELY OR PROPHYLACTICALLY?

The second part of this question did not arise, as the first was answered in the negative. Selecting the badly growing tumours, and subculturing with these, we found that no permanent strain of a badly growing variety could be raised, that is to say, no attenuation was induced in virulency by this process of selection. The variations in growth under inoculation must be ascribed to differences in the self-protective capacity of different mice; or to chance differences in the quality of the grafted material—the proportion of necrotic tissue, for example—inserted with the vigorously growing.

WILL THE TREATMENT OF THE PULP WITH NORMAL HUMAN BLOOD SERUM INHIBIT THE GROWTH OF THE GRAFTED TUMOUR?

The addition of normal human blood serum, pipetted off after spontaneous coagulation, to the tumour pulp, proved to be devoid of any cytocidal action, the growths being well declared on the fifteenth day after subcutaneous insertion. The serum was allowed to act on the tumour pulp for forty-five minutes; the excess was then pipetted off, and the pulp injected subcutaneously.

WILL THE ADMIXTURE OF MOUSE SPERM WITH THE PULP, SEEING THAT THE TISSUE OF A CARCINOMA MAY BE GAMETOID, OR CONTAIN REDUCED CELLS, ACCELERATE THE GROWTH OF THE TUMOUR?

The admixture of sperm with vesicular secretion, to the tumour pulp, neither accelerated nor retarded the growth of it. By the thirteenth day the six mice inoculated had grown oval tumours averaging 1·8 cm. in chief diameter. The sperm used was obtained by lightly etherizing the mouse, almost decapitating from the dorsal aspect, allowing the carcase to bleed in warm water; and then, after washing in alcohol, 5 per cent. carbolic acid, and again in alcohol, by

opening the abdomen, and removing the testicles with the vesiculæ seminales. From the testicles, whilst in salt solution, the epididymes were cut away, and the sperm obtained by dividing the globus minor and expressing the milky contents, which invariably contain numbers of active spermatozoa.

Mouse sperm obtained in the way just detailed, and mixed with the contents of the vesiculæ seminales, exercised, we found, neither a stimulating nor an inhibitory effect when injected subcutaneously on the side of the abdomen opposite to that of a growing grafted tumour. The mice selected were those in which the growths were of small size, the latter being only of eleven days' age. By the end of twenty-one days from the date of the original inoculation, the tumours had advanced to the full, normal size.

A mixture of sperm and of the vesicular secretion, prepared as already described, was found to produce neither stimulating nor inhibitory effect, when injected subcutaneously two days after the subcutaneous insertion of tumour pulp on the other side of the abdomen, the injections being twice repeated. Within fourteen days the growths were the size of peas. The subcutaneous injection, sperm and vesicular secretion (freshly prepared for the occasion), was repeated on the sixteenth day, and again (freshly prepared), on the nineteenth day. The tumours steadily progressed, and were continuing to do so at the end of four weeks, when the animals were killed.

(*December* 5, 1916.)

Feeding Experiments made upon Mice, with Mouse Cancer.

By S. G. Shattock and L. S. Dudgeon.

De laesionibus quibusdam in Muribus repertis qui Murium carcinomata ut cibum sumpserant.

Summarium.

Inceptum est experimentum mense Maio, 1914, quattuor muribus selectis quibus interdum dabantur murium tumores ab illo tumore propagati qui numero "63" distinguitur (Imperial Cancer Research).

(1) Mense Martio, 1915. E quattuor muribus unus qui sumpserat quadraginta duo tumores mortuus est. Neoplasma magnitudinis insignis in thoracis mediastino post mortem repertum est, quod scrutatio microscopica demonstrat esse sarcoma e cellulis rotundis constructum. A pulmone projiciuntur noduli pauci et minuti in structurâ penitiori illi neoplasmati similes.

(2) Mense Aprili, 1915. E quattuor muribus interfectus est secundus cui dati erant quadraginta duo tumores. Vaginae circum extremitatem inferiorem apparuerat tumor in structurâ microscopicâ endothelioma malignum.

(3) Mense Julio, 1915. E quattuor animalibus interfectus est tertius cui dati erant quinquaginta tumores, quod abdomen tumuerat graviditate absente. Post mortem repertae sunt abdominis intra cavitatem duae moles, telae autem solum adiposae, quae uteros circumdabant, et quarum originem explicare oportet mure gravescere cessatâ.

(4) Mense Augusto, 1916. Mortuus est mus quartus cui dati erant septuaginta duo tumores. Post mortem inventus est nodulus in pulmonis superficie inclusus, organo nihil aliud morbidi monstrante. Hunc nodulum scrutatio microscopica neoplasma esse demonstrat e cellulis epithelii vesicularum ortum: inflammationis notae totaliter absunt.

De his observationibus dicere licet. Neoplasmata quum inter se differant et quum in structurâ dissimilia sint eis tumoribus qui per os ut cibum sumpti erant, non explicari possunt cellulis implantandis.

Hypothesem alteram parasiticam tamen proponere licet: tumorum esorum cellulae forsitan includunt contagium, etiamsi ultramicroscopicum, quod postquam in intestino liberatum atque absorptum est, partem corporis inficere potest si quas cellulas conjugatione sive symbiose aptas inveniat.

The first systematic attempts in this country to infect the lower animals with cancer by feeding, were those carried out by one of us (S. G. Shattock) in conjunction with Mr. C. A. Ballance.[1] These were made upon white rats, which were fed from time to time with mammary carcinoma recently excised from the human subject. As noticed in a more recent paper,[2] such an experiment would involve a heteroplastic grafting of cells, and its success as one may now see, is from this standpoint, open to adverse criticism. In the present experiment, mice were fed for long periods upon mouse cancer, with results which, we venture to think, are worthy of consideration. In the paper last referred to, amongst certain introductory observations upon the pathogenesis of carcinoma, it is said: "Whether the disease is *infectious* in the more restricted clinical sense—i.e., conveyable from one individual to a second without personal contact—is still a question that cannot yet afford to be ignored. Guelliot[3] in an analysis of seventy-seven cases in which a transference suggested itself, points out that seventy-one were man and wife; and six, persons living together, masters and servants, parents, &c. In nineteen of this total the disease affected similar organs (stomach eleven, mouth four, skin two). In fifty-eight it affected dissimilar parts. It has been urged by some that the latter fact disposes of the possibility of the disease having been transferred. This is not, however, the case. The result can be viewed as due, not to the implantation of the living cells under such circumstances, but to the access and different distribution of a hypothetical symbiobe: the cell may be dead without the virus being so, or the virus may have been liberated."

The theoretical indications suggested by the results of these feeding experiments are discussed at the end of the present communication; they re-open the question of a parasitic ætiology of cancer as rehabilitated in the form of an ultramicroscopic virus symbiotic with the cell.

The feeding was commenced on May 25, 1914, four normal mice,

[1] *Proc. Roy. Soc.*, 1890, xlviii, p. 392.

[2] S. G. Shattock and L. S. Dudgeon, *Proc. Roy. Soc. Med.*, 1915, viii (Sect. Path.), p. 5.

[3] *Gaz. des Hôpitaux*, 1892, lxv, p. 1209.

not fully grown, being selected for the experiment. The earlier progress of the observation is noticed in the paper already mentioned, at which date (December 31, 1914) the results were negative: the animals had then received thirty-three tumours, and had borne many litters of young, three of the four being females. For the supply of tumours, mice which had been subcutaneously inoculated were, from time to time killed, the tumour being afterwards removed and pinned to a slip of wood. The animals were fed in addition with oats, carrot, boiled potato, &c. The tumour used throughout was that known as No. 63, at the Imperial Cancer Research; none were given in which any trace of ulceration had occurred; they were eaten with avidity.

No. 1: *March* 25, 1915.

One of the series, a female, was found to have died without having been obviously ailing, or ill-nourished. It had received forty-two tumours; the last, on March 6. The experiment had extended over a period of ten months.

Post mortem: A complete autopsy was carried out. Abdomen: The stomach and whole of the intestine were slit up and swept out with a soft brush; there was no growth either in the stomach or anywhere in the intestine. From the peritoneal aspect of the small gut, there projected two hemiovoidal nodules, nothing indicative of the presence of which appeared on the mucosal aspect—these being lymph knots, which in the normal mouse exhibit the same subperitoneal prominence and are sparsely distributed on the small intestine; the muscular coat invests the nodule on the peritoneal aspect. Liver, spleen, kidneys, normal. A careful dissection was made of the tongue, mouth, pharynx, larynx, and of the œsophagus and trachea in the neck; there was no new growth in any of these positions. On opening the thorax, the attention was at once arrested by an extensive growth filling its upper part, and associated with the projection into either pleura of a lobulated mass prolonged from its lower aspect, and having the same microscopic structure. In the mesentery there was one notably enlarged lymphatic gland.

Microscopic examination: The sections were cut horizontally and made so as to include the whole of the thoracic contents, with the exception of the lungs. On one side of the trachea, at its point of bifurcation, there are three lymphatic glands lying in the mediastinal fat, enlarged from the same histological change as that to be presently described. The rest of the mass consists of fat in varying degrees of

replacement by a new formation, the replacement being, in large areas, complete. If the lesion has arisen in the remnants of the thymus, it is not limited to these, for the infiltration is posteriorly quite undefined and involves the connective tissue behind the great vessels to which it is, moreover, adherent. Whether the formation has arisen in the tissue representing the thymus, or in the mediastinal lymph glands is, however, immaterial. The cells include a certain number of lymphocytes, but they are chiefly of the connective tissue type with large nuclei having an open chromatin net, and do not correspond with plasma cells (Unna). They are closely packed without any alveolation, and they nowhere exhibit any indication of fibroblastic development. In two comparatively small areas necrosis has occurred. The total absence of giant cells excludes a tubercular pathogenesis. In the necrotic areas chromatolysis is abundant. Even in young regions of the infiltration, where necrosis is quite wanting, chromatolysis is to be recognized; and it is almost invariably limited to the small, round, deeply stained nuclei of the lymphocytes, which throw out minute processes, sometimes pyriform, from the periphery, and subsequently break up into deeply staining molecules. The changes in the three enlarged lymphatic glands first referred to, are identical with those in the mediastinal fat. And sections of the lobulated mass projecting into the pleura exhibit the same structure. In the blood within the great vessels there is no excess of polymorphonuclear leucocytes or of lymphocytes, or other uncoloured cells. The formation may be classed as a form of round-celled sarcoma; whether the lymphatic glands about the trachea were infected primarily or secondarily, is difficult to say. This view is strengthened by the structure of the minute pleural nodules to be next noticed. The section carried through the affected portion of the lung shows three such nodules. One of these is a concavo-convex elevation, beneath which the outline of the lung is traceable—i.e., it is epipleural as displayed in the plane of section, the pulmonary tissue beneath being uninvolved. Not far removed there is a second, slightly smaller, and similar elevation which is not altogether epipleural but involves the underlying surface of the lung. This appears, amongst other ways, from the presence of minute particles of black pigment in some of the cells, similar to the particles in certain of the epithelial cells of the subjacent alveoli. There is a third, still smaller nodule in a slight recess of the surface. All these nodules exhibit the same histological structure as the mediastinal formation. The mesenteric gland, which has a diameter of 5 mm., is uniformly enlarged by the diffuse growth

FIG. 1.

A transverse section of the thoracic contents of the first mouse fed with mouse carcinoma, showing a large mediastinal growth extending around the great vessels above the heart; the œsophagus lies intact behind it. (Enlarged twice.)

Sectio transversa thoracis viscerum e mure, numero (1) qui murium carcinomata ut cibum sumpserat. In mediastino monstratur tumor qui vasa primaria supra cor circumdat. (Bis magnificatur.)

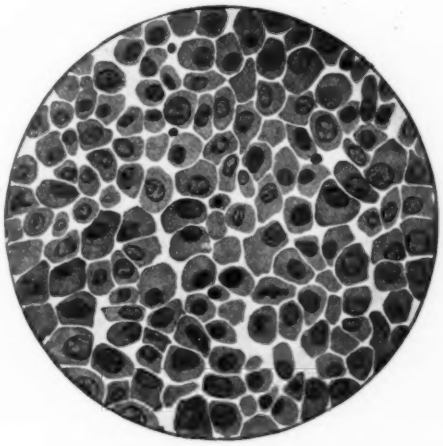


FIG. 2.

A microscopic section of the preceding mediastinal tumour, showing a closely packed formation of cells, mostly of connective tissue type, without alveolar disposition, and exhibiting no fibroblastic development. ($\frac{1}{12}$ obj.; traced with the camera lucida.)

Tumoris praecedentis sectio microscopica. E cellulis, haud lymphocytis, compactis constat sine dispositione alveolari, et nusquam in cellulas fibroplasticas transmutatis.

of a similar round-celled neoplasm, scattered through which there are foci and strands of the original lymphocytic structure of the gland; the small size and deep stain of the nuclei of the lymphocytes contrast with those of the proper cells of the new formation. There are nowhere any fibroblasts in the section, which takes in the entire gland; neither are there any giant cells, nor necrosis. The presence of an adjacent fragment of normal pancreas, shows that the gland lay in the root of the mesentery.

No. 2: *April* 1, 1915.

A second mouse (female) was killed, as it had developed a prominent tumour, as large as a haricot, in the right groin, and projecting into the right side of the perinæum. The animal had received forty-two tumours.

Post-mortem: On reflecting the skin, which was thinned but not infiltrated, a somewhat firm, lowly lobulated tumour, blotched of a deep red, was exposed: it lay on the right of the lower end of the vagina and closely around it, but without involving its wall, or projecting into the canal. The rectum on dissection proved to be quite intact. The inguinal lymphatic glands which lay in close connexion with the inguinal extension of the mammary gland were not enlarged, and microscopically were quite normal in structure. The pharynx, larynx, and œsophagus were normal, as were likewise the lungs, and the several abdominal viscera comprising the uteri, liver, spleen, pancreas, and kidneys. There was no intestinal obstruction, and no growth within the stomach or the intestines. Projecting from the exterior of the small intestine, there were a few prominent hemiovoidal nodules, similar to those described in the first mouse, and which microscopic section, as in that case, proved to be lymphatic.

The new growth is, microscopically, uniform in its construction, and is imperfectly parted into lobules by strands of connective tissue; hæmorrhagic areas are scattered through it. At its periphery the neoplasm is minutely invading the surrounding fat in the manner characteristic of a malignant formation. As studied in the advancing edge it proceeds by the extrusion of narrow, somewhat cylindrical, groups of cells which become later on canalized by the formation of a homogeneous material. None of the lumina contain blood; and hence it cannot be regarded as an angiosarcoma or malignant form of perithelioma. The cell masses may, however, be more voluminous and consist of multiform elements of which the peripheral are

subcolumnar and have a palisade disposition; and in place of exhibiting only a single central lumen, present multiple spaces filled with similar substance, and bounded by a single layer of cuboidal or subcolumnar cells; these channels, like those first referred to, result from the canalization of solid cords, the cells of the general mass immediately around becoming somewhat flattened from the resulting pressure. The distension may proceed to the production of microcystic channels filled with a vacuolated homogeneous substance; under which circumstances the cells bounding the spaces become secondarily

FIG. 3.

The tumour (of which a slice has been cut away) in the second mouse fed with mouse carcinoma, shown *in situ*, surrounding the lower part of the vagina, the canal of which, at one spot, is slit open. A bristle has been passed into the vaginal orifice from the vulva, beneath the clitoris; and another through the anus out by the rectum, which lies behind the tumour and is uninvolved. The neoplasm probably arose in the mamma, which in the mouse, extends into the groin.

Viscerum in pelve inclusorum dissectio e mure, numero (2) qui murium carcinomata ut cibum sumpserat. Vaginae circum extremitatem inferiorem ortus est tumor quem scrutatio microscopica demonstrat esse endothelioma malignum sive sarcoma plexiforme. Neoplasma in mammâ probabiliter ortum est.

flattened. The stroma of the tumour is of the ordinary connective type. No mammary tissue nor lymphatic was encountered in the section, which included the whole thickness of the new formation.

The invaded fat is in places the seat of a small-celled infiltration. The neoplasm may be classed, therefore, as an invasive endothelioma or plexiform sarcoma.

It may be observed in passing, that the tumour differs histologically from that which was used throughout the experiment for the purpose of feeding; as well as in the entire absence of necrosis which invariably occurs in the latter.

FIG. 4.

A microscopic section of the preceding tumour. It presents the structure typical of an endothelioma; at its margin the surrounding fat is being invaded by extensions of the neoplasm, showing that the latter is of an invasive or malignant kind. ($\frac{1}{6}$ obj.; traced with the camera lucida.)

Tumoris praecedentis sectio microscopica structuram monstrans endotheliomatis propriam; tela adiposa neoplasmatis processibus invaditur.

No. 3: *July* 31, 1915.

The third mouse was killed, since the abdomen was very large. The enlargement had persisted too long to represent a pregnancy, although the animal had been many times pregnant by the male of the four with which the experiment started. In this case the experiment had lasted fourteen months, and fifty-one tumours had been used.

Post mortem: The abdominal enlargement proved to be due solely to the growth of two voluminous, symmetrical masses of fat, connected with the uteri; there was likewise a considerable amount of fat in the mesentery. All the viscera were normal, the gut in this case, however, was not critically examined. The accumulation of fat may be here related to the close of the reproductive function, and is to be observed under such circumstances in cases when no superadded factor is in question.

No. 4: *August* 30, 1916.

The fourth and last of the mice (male) died. It had received no fewer than seventy-two tumours, having been so fed for two years and four months.

Post-mortem: On one lung there was a small solid circular area, lowly convex, though scarcely raised above the general level, and measuring 3 mm. in diameter. A more minute nodule, of a dull white colour, about the size of a pin's head, on the upper surface of the liver, proved on microscopic section to be a fibrous sac with calcified contents —obviously some form of obsolete parasite. The stomach and intestine presented no internal growth. On the outer surface of the small intestine, 4 cm. from the stomach, there was a prominent hemiovoidal nodule, 3 mm. in larger diameter; a second and similar projected from the small intestine some way distally to this; these, as in the preceding cases, consisted of normal lymphatic tissue. The testes, bladder, and vesiculæ seminales were all healthy.

Microscopical examination of the pulmonary nodule: This in sections cut vertically to the free surface appears as a sharply circumscribed circular formation of compact, uniform structure. The pulmonary alveoli immediately around are flattened from pressure, but are otherwise unaffected. The nodule is composed of a compact mass of cells, parted in places into groups by narrow meandering fissures. The source of the cells is traceable to the alveolar epithelium; the elements are polyhedral, of comparatively large size, and furnished

FIG. 5.

The nodule in the lung of the fourth mouse fed with mouse cancer. (Enlarged twice.)

Pulmonis portio e mure numero (4) qui murium carcinomata ut cibum sumpserat, nodulum sub pleurâ monstrans. (Bis magnificatur.)

FIG. 6.

A microscopic section of the margin of the preceding nodule in the surface of the lung, showing compression of the surrounding alveoli by the growth, which consists solely of epithelial cells; the source of these is traceable to the pulmonary epithelium. ($\frac{1}{6}$ obj.; drawn with the camera lucida.)

Tumoris praecedentis marginis sectio microscopica. E cellulis epithelii compactis constat. Pulmonis vesiculae tumorem circumjacentes compressae sunt. Oritur neoplasma e vesicularum epithelii cellulis.

with oval nuclei; none are fibroblastic, and there is not a single polymorph, lymphocyte, or plasma cell amongst them. The absence of leucocytes and the sharp circumscription of the proliferating process show that the nodule cannot be viewed as a broncho-pneumonic production. The alternative view is that it represents a primary epithelial neoplasm.

In the histological collection of one of us (S. G. Shattock) there is a section of a human tumour, the size of a pea, which was found unexpectedly in the lung at a post-mortem examination, and which is structurally identical with the foregoing; and like it may be regarded as a primary neoplasm arising from the epithelium of the pulmonary alveoli. It consists of compact groups of cells resembling those of the pulmonary epithelium, filling circumscribed spaces the walls of which are mapped out by a capillary plexus distended with blood, and obviously that of the alveolar walls. There is a complete absence of small cells, and there is no pigment contained within any of the cells of the new formation. The intra-alveolar location of the cells of a carcinomatous growth in the lung does not, of course, in itself prove that the new formation has arisen from the pulmonary epithelium. Secondary or metastatic carcinoma of the lung assumes two very different macroscopic forms. The usual form is the nodular or circumscribed; the rarer, a diffuse infiltration of the interalveolar tissue. There are but two specimens of the latter in the Museum of the Royal College of Surgeons, which may be cited in passing, and which the descriptions in the new catalogue will sufficiently explain.

2037.1: Portion of a lung showing metastatic growths of an unusual character, following carcinoma of the breast and of the lymphatic glands of the axilla and neck of the corresponding side. The new formations take the form of fine opaque, branching and moniliform lines, which at the uncut external surface of the lung appear in places as a plexus. The growth, as studied in the section, closely accompanies the blood-vessels, and probably lies in the perivascular lymph spaces. The liver was enlarged, and was the seat of secondary tumours, the larger of the superficial of which were umbilicated.

2038.1: A slice of lung showing its infiltration with carcinoma, secondary to disease of the pelvic colon. Its substance is everywhere strewn with minute semitransparent foci, which are sections of perivascular and peribronchial lymphatics, dilated with new growth in process of mucinoid degeneration. Histologically the infiltrations consist of polyhedral cells, much distended and finely vacuolated with

mucinoid material; the cell groups are devoid of lumen. From a girl, aged 15, suffering from carcinoma of the pelvic colon. On the abdomen being opened, growths were found scattered over the peritoneum; free fluid was present in large amount within the abdominal cavity. A few days later signs of acute obstruction appeared. Colostomy was performed, but the patient died.

The intra-alveolar position of the cells in the nodular form of metastasis is easily recognizable, and was long ago accurately described by Cornil and Ranvier.[1] One of the best ways of demonstrating it is to stain the sections with Unna's acid orcein after the ordinary use of hæmatoxylin; the abundance of elastic tissue so shown in the walls of the spaces at once proves their nature: the section becomes mapped out into a regular pattern, which obviously is that due to the distension of the pulmonary alveoli with the neoplastic epithelium. The constancy with which this can be demonstrated indicates that the carcinomatous cells, though transferred to the capillaries, rapidly break through in their growth, and extend into the alveoli; a result attributable to the extreme tenuity of the tissue composing the alveolar wall, the vessels of which are covered directly with the pulmonary epithelium.

Remarks.

In discussing the results of these feeding experiments, the intestinal lymph nodes may be ignored; they are neither enlarged nor structurally altered; and there is no evidence that they represent entrance sites of infection, or anything having the nature of a specific anatomical sign of malignant disease.

The first striking thing is that the tumours in the three mice have not the same histological structure; and that none can be viewed as having resulted from the growth of implanted cells, since none repeat the histological characters of the tumour used for the purpose of feeding (viz., No. 63, Imperial Cancer Research).

Thus, the first is a round-celled sarcoma of the mediastinal lymphatic glands and mediastinum, and of a mesenteric gland near the pancreas: the route of infection here may have been by way of the intestine or of the respiratory track.

The second is an invasive endothelioma surrounding the lower end of the vagina, and probably arising in the inguinal extension of the mamma. And it differs from that used for the purpose of feeding,

[1] "Manual of Pathological Histology," 2nd ed., 1880.

amongst other ways, in the complete absence of the necrosis which invariably occurs in the latter.

The third is an uncomplicated epithelial neoplasm of the lung arising from the alveolar epithelium.

In the numerous examples of cancer occurring in husband and wife, the same diversity is witnessed, with a few exceptions; and so it is among the tenants of "cancer houses"; whence the same inference must be drawn—viz., that the tumours cannot be due to the growth of implanted cells. Equally true is it that the tumours arising in the members of cancerous families seldom affect the same organ and have a similar histological structure. Thus Paget[1] states that he "saw a young lady, aged 24, who had epithelioma of the pharynx and quickly died with it. Her mother, grandfather, and great-aunt and great-grandmother (all in one line) died with cancer of different organs." Amongst other instances adduced by Paget (loc. cit.) are: (1) A man had medullary cancer of a toe, his father had cancer of the lip. (2) A woman had repeated epithelial cancers of the labia: her sister, her father's sister, and her mother's brother's daughter, had cancer of the breast. (3) A man had epithelial cancer of the lip, whose grandmother had cancer of the breast. (4) A gentleman had epithelial cancer of the interior of the cheek; his aunt died with cancer of the breast. (5) A woman had medullary cancer of the breast; her mother had cancer of the uterus, and her uncle cancer of the face. (6) A woman had scirrhous cancer of the breast, whose mother's uncle had cancer of the lip. (7) Of another woman with similar cancer, one cousin had cancer of the lip, another cousin cancer of the uterus. (8) A third woman had scirrhous cancer of the breast, whose grandfather had cancer of the lip. And he cites from Broca ("Traité des Tumeurs") the case of a woman who died of cancer of the breast, of whose married daughters, two died with cancer of the liver, and two with cancer of the breast; one of these daughters had seven children, of whom one son died with cancer of the stomach, three daughters with cancer of the breast, and one with cancer of the liver; another of the daughters had also seven children, three of whom died with cancer of the breast, one of the liver, and one of the uterus; and of those daughters, one had a daughter who died with cancer of the breast. Dr. Herbert Spencer tells of a family in which a lady died a few weeks after parturition, with a sarcomatous

[1] "Surgical Pathology," 3rd ed., 1870, p. 793.

tumour of the left ilium; and whose two brothers died later with abdominal sarcoma.

The simultaneous growth of two independent carcinomata of different histological structure is an occurrence akin to this, and one of which a large number of examples can be brought together. What is more, in cancerous families the malignant growth may in some members take the form of a sarcoma. To quote from the same authority: "In hereditary transmission the cancer material may be modified so that the form of the disease in the offspring may be different from that in the parent. The change from scirrhous to the medullary cancer, and vice versa, is not rare. I have mentioned cases of alternation between those and the epithelial cancers; and a case of melanoid cancer in a patient descended from one with a scirrhous breast." It may be pointed out here that Paget uses the word cancer to include all malignant tumours, whether carcinomatous or sarcomatous, and that medullary cancer might, therefore, be a soft form of either. The melanoid cancer, however, may safely be taken as having been a melanotic sarcoma. In this last connexion, may be cited the cases in which a carcinoma and a sarcoma occur in different organs of the same individual, or even in the same organ. A fair number of examples of the last association have been recorded in the uterus. Dr. Herbert Spencer has described one such, and collected others,[1] and one has lately come into the College possession through Dr. J. P. Hedley, in which the uterine cavity is distended with a sarcomatous tumour, whilst the endometrium is the seat of an ordinary carcinoma.

And as a still closer connexion we have the occurrence of the mixed form of neoplasm, the carcino-sarcoma, where at the same spot both manifestations of malignancy, epithelial and connective tissue, are intermingled.

There is, however, a second hypothesis of transference as a post-natal phenomenon, which the various facts brought together suggest, viz., that the cell may be the vehicle of an ultramicroscopic virus symbiotic with it; and that if transplantation of the cell fails, the virus may be freed, and after its absorption and distribution may infect some structure of a second individual, provided that there is any such structure prepared for its symbiosis: from the latter reservation, there is logically no escape.

[1] *Obst. Soc. Trans.* 1905, xlvii, p. 338.

In some such way the production of carcinomatous disease might follow in another organ than that originally affected, and the resulting lesion present a different histological structure. Whether the virus, in hereditary transmission, is itself transferred, or whether the transmission is limited to the aptitude of certain cells for infection after birth is a matter that need not be discussed. But it will be clear that the parasitic hypothesis is not disposed of by the facts established in regard to heredity. The problem is, in this respect, parallel with that of tuberculosis where the phenomena rarely concern the transmission of the bacillus, but the aptitude of particular members of a family to receive infection. As in other bacillary infections, the virulence of the micro-organism and its dosage, naturally play important parts; nevertheless, the factor of predisposition is one recognized by clinicians; and means, that given the same virulence and the same dose, some individuals would be infected whilst others would escape. The same factors would, all of them, obviously obtain likewise in the case of cancer, supposing it were due to an extraneous infection. Those living with, or nursing cancerous patients, would be subjected to greater exposure, and so be more likely to contract the disease. Nor is the incidence of "spontaneous" cancer amongst mice kept together in confinement surprising on such an hypothesis: a cancer laboratory is, in fact, a cancer-house.

And somewhat the same answer may meet the objections raised by the results of the experimental prophylactic immunization against grafted mouse cancer, by the previous subcutaneous injection of mouse blood, or of mouse embryo, or mouse skin: they so modify the fate of the grafted cells that the virus is rendered harmless, since no other cells are prepared for its symbiosis.

If the results of the mouse-feeding experiments recorded in the present communication are to be interpreted, then, on a parasitic hypothesis, it would be that the carcinogenic virus is freed, on the destruction of the ingested cells, and transferred after absorption, to the body of the host, wherein it incites the formation of a malignant neoplasm in some spot where the cells are prepared for its symbiosis.

DISCUSSION.

Dr. J. A. MURRAY: I may point out that the round-celled growth and the tumour of the lung are conditions common in the mouse, and of doubtful malignancy. If these two growths are excluded for these reasons, one mouse in four had developed cancer under the conditions of the experiment, and this is not an exceptionally high proportion for mice of 2 years of age and over. The results do not, therefore, in my opinion necessitate the assumption of the transference of a virus from the tumours with which the mice were fed, to the cells of the animals eating the tumours.

Mr. MCADAM ECCLES: I have been much interested in the report of these experiments of feeding mice with "mouse cancer," and in hearing that Mr. Shattock is inclined to revert to the parasitic theory of the origin of malignant tumours. I confess that I have never wavered from such a belief, and the reference to the old idea of "cancer-houses," reminds me of some facts which concerned me in my youth because of their connexion with my own family. There was an old Georgian farm-house in South Devon, lying somewhat low, and surrounded by hills, with a stream running by one side and passing into a pond below the level of the front entrance. The house had several large cellars beneath it, which were always damp. All its rooms, but particularly the kitchen and the servants' quarters, were infested with mice. The farm and its buildings had been for many years in the possession of my family, but the house was usually occupied by tenants, who were not members of the family. Among these, several instances of malignant disease having been the cause of death were known, although I have unfortunately no details. Some forty years ago a member of my family and his wife, a lady several years older than he, came to occupy the house. My family on this side is a long-lived one, and has been altogether free from malignant disease for at least two previous generations. Some eight years after the above (who had no children) entered into possession, one of the elder female servants developed carcinoma of the mammary gland; and two years later the wife contracted carcinoma of the larynx and died in the house. About three years afterwards the husband, then aged 40, who had remained on in the house after his wife's death, became the subject of primary carcinoma of the liver. The house was subsequently tenanted by another family, of whom two members died within its walls from malignant disease; details of these cases I cannot furnish. The house has since been demolished. From a purely clinical point of view, it has always seemed to me that malignant disease was in some way connected with an infective agent; and I have often wondered whether the domestic mouse, always with us and frequently fouling our food, might not be a carrier or the intermediate host of a virus capable of producing sarcoma or carcinoma in the human subject.

Section of Pathology.

President—Dr. F. W. Andrewes, F.R.S.

(*February* 13, 1917.)

The Changes in the Central Nervous System in Hypothyroidism.

By F. W. Mott, Major R.A.M.C., M.D., LL.D., F.R.S.

I have published two communications in the *Proceedings* of the Section of Psychiatry of this Society on the changes in the central nervous system in hypothyroidism. The nervous symptoms of myxœdema point to a condition of exhaustion of nervous energy, which can be remedied by the administration of thyroid gland; consequently *a priori* upon the assumption that the basophile resistance of the nerve cell is a source of nervous energy, we should expect to find what actually I have now found to exist in seven cases—viz., a marked chromatolysis, which in sections of the brain and spinal cord is revealed by the partial or complete disappearance of the Nissl granules throughout the central nervous system.

Since the former publications, a very advanced case of hypothyroidism was admitted to Claybury Asylum: this patient showed well marked signs of myxœdema and very marked mental nervous symptoms. Before recounting the clinical notes and pathological findings in this case, which accord in great measure with the other cases recorded, except that the mental symptoms were more profound, I will briefly relate a summary of the clinical facts and pathological findings in the six previously recorded cases. But before doing so, I should like to call attention to a valuable paper by Dr. Kojima on the

"ductless glands in 110 cases of insanity." This work was done in my laboratory, and published in the *Proceedings* of the Section of Psychiatry of the Royal Society of Medicine (vol. viii). I shall have occasion to refer to one or two conclusions at which Dr. Kojima arrived in connexion with hypothyroidism and the other ductless glands.

Conclusions regarding a Correlation of Hypothyroidism and Changes in the Central Nervous System.

There is a universal chromatolytic change in the cells of the central nervous system, sparing no system or group of systems of neurones entirely. The changes have been especially noted in the smaller cells of the autonomic nuclei—e.g., the vagus and glosso-pharyngeal. The cells of the olivary body show much less change, and the Purkinje cells of the cerebellum show less chromatolysis than the large motor cells of the medulla oblongata, spinal cord and Betz cells of the cortex, and there is no increase of neuroglia. All the asylum cases, especially four of the five, showed marked chromatolysis of the cortical cells. The one that showed the least degree of mental confusion and other signs of insanity, exhibited the least change in the cortical cells.

The lantern slides of the case I am about to show exhibit very marked changes, and the mental symptoms in this case were most pronounced. Unfortunately, the notes do not permit correlation of the changes in the bulb, in these asylum cases, with the symptoms indicative of affection of the autonomic nuclei, as was possible in the hospital cases.

But I have come to the conclusion that there is a type of insanity in women about the climacteric, in which a manic-depressive condition, associated with mental confusion, hallucinations, delusions—mainly of persecution—loss of memory of recent events, slowness of thought and utterance, and terminating in dementia, may arise as a result of a particular form of hypothyroidism. This hypothyroidism is characterized:—

(1) By an atrophy of the glandular structure of the thyroid, interstitial fibrous hyperplasia, and abundant infiltration of the same with lymphocytes; a condition of chronic inflammation arising from a toxic condition, probably local in its source, as Kojima has shown; the adjacent parathyroids do not exhibit this change.

(2) By an increase in weight of the pituitary gland as a rule, but not always; nevertheless, there is, as was shown by Boyce and Beadles

long ago, and later by Herring in experimental hypothyroidism, an increase of the *pars intermedia*. Herring has also shown that the cells of this structure invade the *pars nervosa*, and I shall show that this has occurred in the case under consideration. This is of interest, for Schäfer, in his work "The Endocrine Organs," states: "Whether the phenomenon also occurs in myxœdema has not been noted, but it will probably be found to be the case." All five cases of hypothyroidism, in which the pituitary was examined, showed an excess of colloid in the *pars intermedia*. As a control it may be stated, that the characteristic perinuclear chromatolysis of the nerve cells found in these cases was not discovered in two cases of simple atrophy of the thyroid gland. Examination of the thyroid gland in these two cases showed still normal colloid vesicles, and although there was an increase of fibrous tissue, there was no lymphocytic infiltration, indicative of a local toxic inflammation; neither was there any increase of the *pars intermedia* or colloid material, observed by Dr. Kojima.

Of course we have always to consider how far the atrophy of a gland, and failure of its function, may act in upsetting the bio-chemical balance existing between the secretions of all the ductless glands and the sexual glands. In the five asylum cases in which the suprarenal glands were examined a deficiency of lipoid in the cortex was observed, but it would be unscientific to correlate this condition certainly with the thyroid atrophy, for the patients have died usually of some acute infective disease, and Elliot has shown that the lipoid is diminished in cases of death from infective disease. Still it is suggestive. Dr. Kojima, in his exhaustive examination of all the ductless glands in three cases, came to the conclusion that he could exclude the probability that changes in any other ductless glands than the thyroid can be responsible for the nervous and mental symptoms, and the histological changes in the central nervous system. It is suggested, however, that the ovaries were affected in three cases. "It may be remarked that these were the cases in which there was not merely a glandular atrophy, but there was also a marked chronic inflammatory, interstitial change."

This case, which I am about to show, had extraordinarily large ovaries and an enormous number of corpora albicantia, but healthy Graafian follicles were present.

It may be mentioned that in all these cases the blood and cerebro-spinal fluid gave a negative Wassermann reaction.

A Case of Myxœdema.

E. A., aged 38. Admitted to Claybury Asylum on March 23, 1916, with no history of previous illness or treatment. The medical certificate states: "She is continually shouting and screaming out that she is falling."

"*Physical Examination.*—Bruises were found on forearms and legs. The muscular system showed paresis. The abdomen was distended, and the lungs full of râles. There was incontinence of urine, and continuous metrorrhagia. The knee-jerks were absent, but the patient's condition did not permit of systematic examination of the reflexes, as she was in the last stage of exhaustion when admitted."

Abstract of Notes made on Admission.—"Patient was suffering from confusional insanity with myxœdema. She was at times very noisy, due to hallucinations of sight and hearing. She rambled inconsequently of things she imagined she saw and heard. She thought she was falling through space and was inclined to be noisy. She took nourishment—milk and brandy and beef-tea, but her breathing became laboured, and her face and hands blue. Her pulse was barely perceptible, and temperature subnormal. General weakness and restlessness developed with the onset of broncho-pneumonia. She died from heart failure about thirty-eight hours after admission."

Notes of Post-mortem Examination.—"Examination of the brain showed considerable wasting of the convolutions, with enlarged sulci, especially in the central region; the cortex was thin and the striation poor; the fourth ventricle was not granular, and the membranes stripped without erosion. The weight of the brain was 1,115 grm., the right hemisphere weighing 480 grm. and the left 475 grm. The dura mater was slightly thickened, and there was excess of fluid in the subdural space. The pia-arachnoid was slightly opaque but not adherent. There was slight atheroma of the vessels. The thyroid was very small and atrophic. The bronchial glands were enlarged and black, and the lungs congested and filled with muco-purulent material, and showed signs of commencing broncho-pneumonia; there was adhesion of the posterior border and base of the left pleura. The right lung weighed 655 grm. and the left 610 grm. The heart was well shaped, with a marked deposit of fat; the right ventricle enlarged, as also the auricle, and there was slight atheroma of the aorta and great vessels. The liver weighed 1,470 grm., and was enlarged, with increase of fibrous tissue, and opacity of the capsule in some places. The bile was dark, and the

spleen weighed 145 grm. The kidneys each weighed 160 grm.; they stripped easily, were slightly atrophic, with a fatty deposit everywhere. The adrenals were small, but of normal appearance on section. The uterus was long and in good condition, but the ovaries were unusually large and abnormally long: as long as the tubes. The right ovary weighed 7 grm. and was 3 in. long; the left ovary weighed 5·5 grm. and was $2\frac{1}{2}$ in. long. The patient weighed 68 kilos; her hair was very thin, and her general condition fatty. The muscular system was covered with a fatty deposit, and her teeth were bad. The cause of death was heart failure."

Remarks.—The practitioner under whose care the patient had been told me that he had considered the case was one of general paralysis of the insane. She was demented, and her speech was affected. Seeing that he had had experience as a medical officer at an asylum he was surprised when I told him that the brain presented no macroscopic or microscopic signs of this disease, and that the case was one of myxœdema with confusional insanity.

Microscopic Examination.

Portions of the ascending frontal cortex, the basal ganglia, the pons and medulla, after hardening in formaldehyde solution, were prepared for sections by the paraffin method. Also the pituitary, thyroid and suprarenal glands were similarly treated.

Sections of the brain, 5 c.c. thick, were cut and stained by Nissl's method, also with polychrome and thionin dyes. A very distinct chromatolysis of the cells in all the regions was observed; especially marked was the change in the Betz cells of the cortex, although all the pyramidal cells are more or less affected. Some cells have lost all their basophile staining substance; in others there is a marked perivascular chromatolysis and a complete or partial disappearance of the granules. The nucleus is often eccentric in position (*vide* photomicrographs, figs. 1 and 2).

The cells of the medulla, the pons and the basal ganglia, show a similar marked chromatolysis (*vide* photomicrograph, fig. 3). The changes correspond indeed with those I have previously described as occurring in myxœdema, only they are, generally speaking, more advanced; especially do I refer to the cells of the cortex.

Sections of the *thyroid* were cut and stained with hæmatoxylin and eosin. The characteristic appearance observed in the other cases was

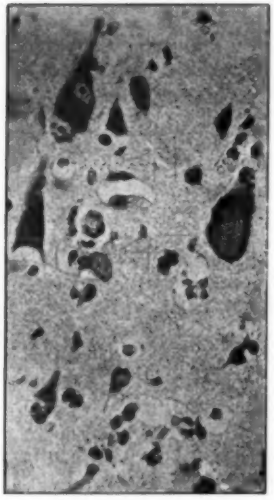


FIG. 1.

Section of frontal cortex, stained by Nissl's method. The small, medium, and large sized pyramidal cells all show advanced chromatolysis. (× 480.)

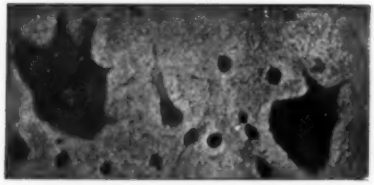


FIG. 2.

Section of ascending frontal convolution, stained by Nissl's method. Two large Betz (psychomotor) cells exhibiting advanced chromatolysis. No tigroid substance can be seen. (× 390.)

demonstrable—namely, almost complete disappearance of the colloid in the vesicles, a marked interstitial fibrosis and lymphocyte infiltration.

The Pituitary Gland.—Sections were cut and stained with hæmatoxylin and eosin, and microscopic examination showed that the *pars intermedia* exhibited an excess of colloid, and an extension of this portion of the gland into the *pars nervosa* to such a degree that outlying isolated cells were observed almost throughout the whole posterior portion of the gland. The examination of the vesicles of the *pars intermedia* with a high power magnification shows that the colloid substance is a secretion of the cells, for their cytoplasm can be seen filled with a hyaline eosin oxyphile colloid staining substance, similar to

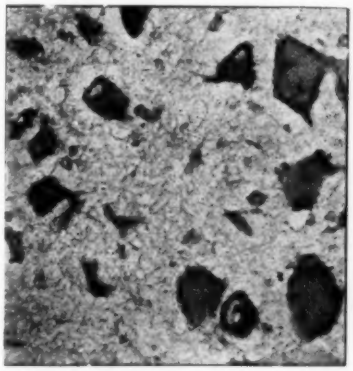


FIG. 3.

Small and large cells of the hypoglossal nucleus showing advanced chromatolysis. (× 340.)

the oxyphile colloid in the vesicles. The most outlying cells in the *pars nervosa* also show this hyaline eosin staining substance, *vide* fig. 4. I have not found "an increase of free hyaline and granular masses" through the *pars nervosa*, described and figured by Herring, as occurring after removal of the thyroids, but the conditions above noted are of a similar nature though less in degree. What part this increase of the colloid of the pars intermedia may play in accounting for the clinical and anatomical changes in the nervous system, it is not permissible to say on existing evidence. It might be argued that it is a compensatory change; Schäfer, however, states "the pituitary colloid

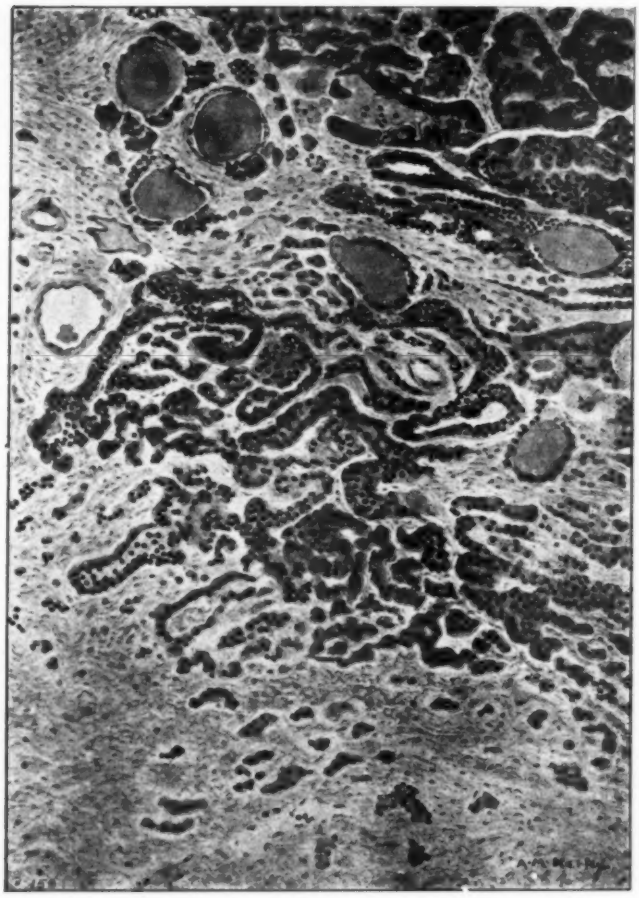


FIG. 4.

Drawing of the *pars intermedia*, showing the colloid in excess, extension of the gland structure into the *pars nervosa*, and outlying isolated cells scattered through the whole of the adjacent posterior lobe. (× 100.)

is not identical with that of the thyroid. It is noteworthy that it does not contain iodine, which is a characteristic component of the thyroidal colloid in all animals. Even many months after thyroid removal, Sutherland Simpson and Andrew Hunter were unable to detect the least trace of iodine in the sheep's pituitary. The pituitary cannot take the place of the thyroid in animals affected with cachexia thyreopriva, nor is pituitary extract able to take the place of thyroid extract in the treatment of goitre and myxœdema. There is therefore no evidence that these two organs act vicariously." However it seems that, as Cushing and Herring have shown that the colloid of the *pars intermedia* passes into the third ventricle, it therefore enters the cerebrospinal fluid, and in this way it may have some influence in determining the nervous symptoms and the chromatolytic changes on the nerve cells. The physiological activity of the posterior lobe of the pituitary gland is connected with the presence in it of the colloid secretions of the pars intermedia, for it is difficult to believe that the neuroglial tissue, of which the posterior lobe consists, can possess active autacoid properties.

The Suprarenal Glands.—Sections were cut by freezing formaldehyde hardened portions of the gland; these were then stained with Scharlach and hæmatoxylin, and mounted in Farrant's solution. These showed marked diminution of the lipoid contents of the cortex, more so than I think can be accounted for by the broncho-pneumonia found post mortem. The *ovaries* were extraordinarily large, contained a great number of corpora lutea vera, and numbers of normal Graafian follicles, pointing to a high degree of reproductive activity; whether this evidence of generative hyperfunction can be associated with the changes in the thyroid gland I am unable to say, but it is well known that an important interaction exists between the thyroid and the generative glands. The thyroid becomes enlarged at puberty, during menstruation and during pregnancy. Myxœdema is much more frequently met with in women than in men.

(*February* 13, 1917.)

Phalangeal Anarthrosis (Synostosis, Ankylosis) Transmitted through Fourteen Generations.

By Dr. H. Drinkwater.

This rare condition is illustrated in the radiograph of the hands of A. T., who very kindly had it taken at my request, and to whom I have to express my grateful thanks. He also supplied me with many of the following particulars. The special scientific interest of the abnormality is the fact—or what is almost certainly a fact—that it has been inherited from an ancestor who died in the year 1453. It thus constitutes the longest period through which an anatomical abnormality has been traced. It is true that the Hapsburg lip has been traced back to the fourteenth century, but this can scarcely be called an abnormality.[1]

Anatomical Features.

Thumb and first finger: These are normal in every respect, and all the joints have the natural mobility.

Middle finger: The terminal joint is freely movable, but the next joint, between the middle and the proximal phalanges, is only very slightly so, and the finger at this part is seen to be considerably swollen, from an enlargement of the articular ends of the bones.

The ring and little fingers: These show the characteristic defect. The distal interphalangeal joint is free in each, but the proximal joint is absolutely immovable, from complete continuity of the middle with the proximal phalanx.

The cutaneous creases and folds of the knuckles are normal over the movable joints, but are absent over the joints that are fixed—i.e., the skin of the ring and little fingers is quite smooth except over the distal articulation.

All the fingers are movable at the metacarpo-phalangeal joints, but the middle, ring and little fingers cannot be made to touch the palm.

Both hands are perfectly symmetrical.

[1] Vide Woods, "Heredity in Royalty."

The feet: All the toes, except the hallux, are affected exactly like the ring and little fingers — i.e., there is a continuity at the site of the proximal interphalangeal joints. There is no other physical defect.

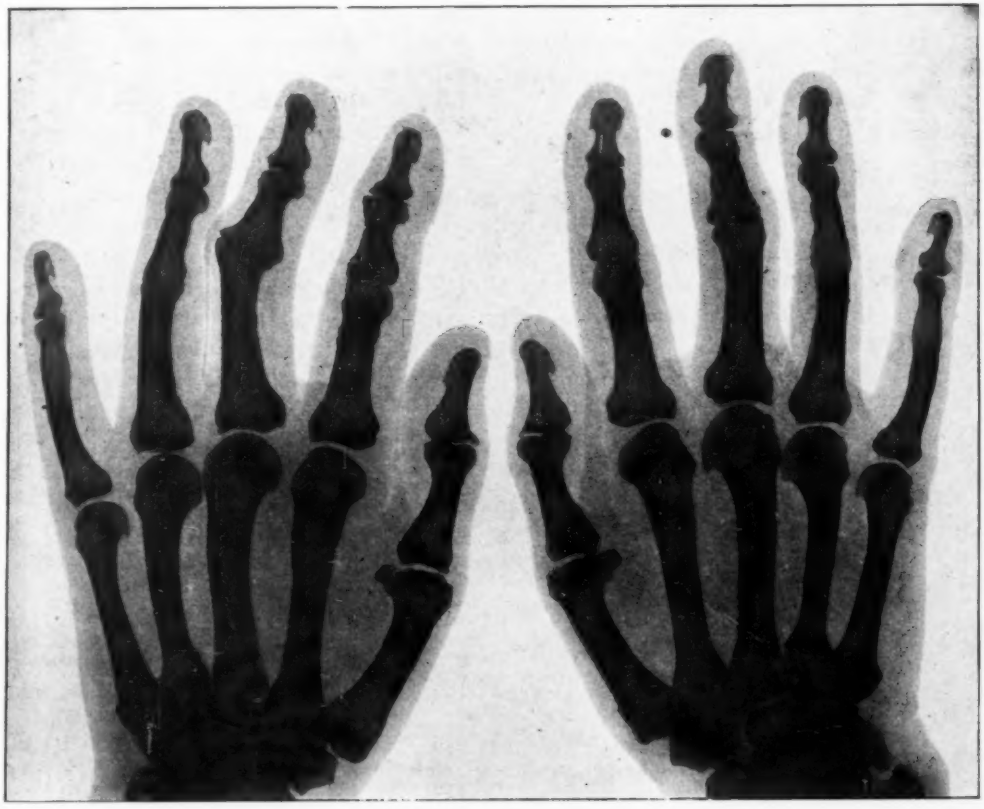


FIG. 1.

A skiagram of the hands of A. T., showing continuity of the proximal and middle phalanges in the ring and little fingers.

A. T. had seven brothers, all tall, the least so being 6 ft. 2 in. in height, which is 5 or 6 in. above the average height of males.

Affected Relatives.

A. T.'s father was married twice; one of A. T.'s half brothers shows precisely the same abnormality, as did also the father and the paternal grandfather; and these are all that were affected so far as he knows for certain.

A. T. is a direct descendant in the male line from John Talbot, the first Earl of Shrewsbury, one of the prominent characters in Shakespeare's Henry VI, and it has been discovered that his fingers showed the same abnormality that I have described in A. T.

John Talbot, first Earl of Shrewsbury was killed at the battle of Chastillon, near Bordeaux, in the year 1453, in which the French were victorious, for with more daring than prudence he attacked the French Army without waiting for the arrival of his Artillery. Tradition said that his thigh bone was broken whilst he was on horseback, and when he fell from his horse he was killed by the blow of a battle axe on the head. He had expressed a wish for his heart to be buried at Whitchurch, because he had fought surrounded by Whitchurch men. His body was brought home and his heart buried immediately in front of the porch. At the side of the porch is a tablet which reads—

> "Beneath this stone (in this porch) rests the embalmed Heart of John Talbot, first Earl of Salop, who for twenty-four years fought his Country's battles against the French and was slain at the battle of Bordeaux A.D. 1453," &c.

His tomb, surmounted by a stone effigy, is inside the church, near the chancel. In 1874 this tomb was repaired at the request and expense of Adelaide, Countess of Brownlow, a descendant of John Talbot, and the skeleton was taken out. When the bones were examined they were found to support the tradition as to the way in which he had met his death; for the skull was found cleft in the right upper occipital region, and the right thigh bone was found to have been fractured.

Bishop Abraham (Assistant Bishop of Lichfield) visited Whitchurch at this time and was shown the bones by the Rector, the Rev. W. H. Egerton, and photographs were taken by Mr. T. R. Crosse, of Whitchurch, who has kindly given me permission to publish these illustrations. Bishop Abraham was a very intimate friend of A. T., and knowing of his digital peculiarity at once examined the bones of the fingers, and discovered the same ankylosis that exists in the fingers of A. T., and he wrote to his friend to inform him of what he had found.

An account of these bones was written by Mr. Egerton, the Rector, and published in the *Standard* in 1877.

In the stone effigy the hands are close together resting on the chest. The fingers are somewhat damaged, but the middle finger shows precisely the same thickening of the proximal interphalangeal joint that is so conspicuous in A. T., as described above. A. T. informs me that

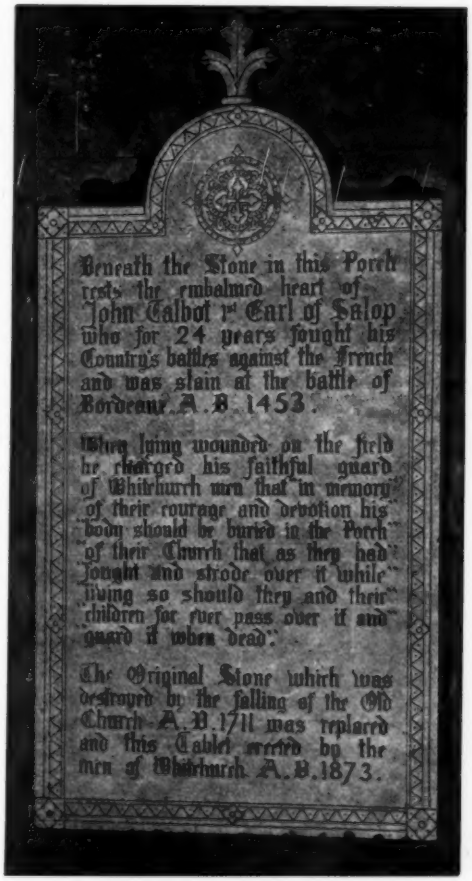

Beneath the Stone in this Porch
rests the embalmed heart of
John Talbot 1st Earl of Salop
who for 24 years fought his
Country's battles against the French
and was slain at the battle of
Bordeaux A.D. 1453.
When lying wounded on the field
he charged his faithful guard
of Whitchurch men that in memory
of their courage and devotion his
body should be buried in the Porch
of their Church that as they had
fought and strode over it while
living so should they and their
children for ever pass over it and
guard it when dead.
The Original Stone which was
destroyed by the falling of the Old
Church A.D. 1711 was replaced
and this Tablet erected by the
men of Whitchurch A.D. 1873.

FIG. 2.

A photograph of the tablet in the porch at Whitchurch, recording the death of John Talbot, first Earl of Salop.

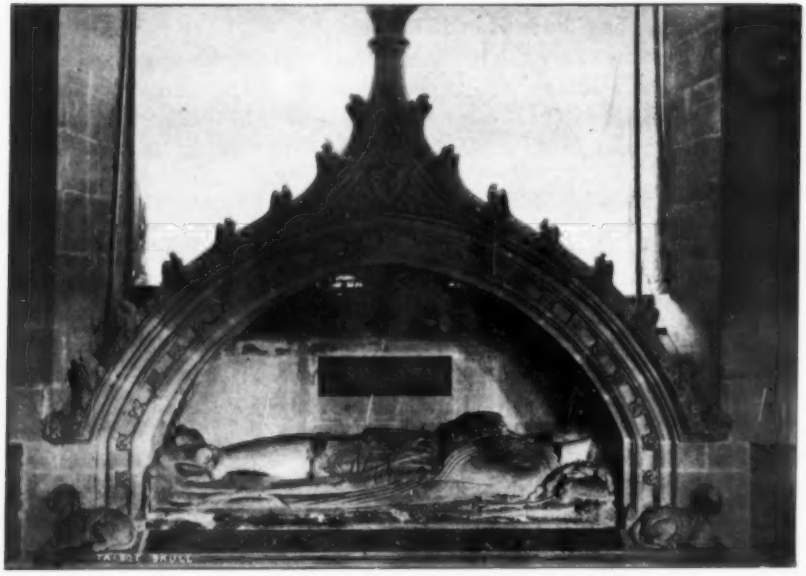


FIG. 3.

The stone effigy of John Talbot, first Earl of Salop, from the tomb beneath which the skeleton was taken out in 1874; the middle finger exhibited the same enlargement as in his living descendant, A. T.

FIG. 4.

The skull of John Talbot, showing a cleft in the occipital region.

"the figure is a contemporary one, so may very well have reproduced John Talbot's finger."

It is clear, then, that the anatomical defect of A. T. has been inherited from John Talbot, who was born more than five hundred years ago, for at the time of his death, in 1453, he was about 65 years of age.

A. T.'s mother and paternal grandmother did not possess this abnormality, nor was it present in the families to which they belonged. The mode of its inheritance in recent members of the Talbot family shows that it behaves as a Mendelian dominant; and, that being so, it must have been inherited by every individual who comes in the direct line of descent from John Talbot to A. T., for a dominant characteristic does not skip a generation.

I have constructed the family pedigree from the data given in "Burke's Peerage," and this shows, in capital letters, the members who would have exhibited this abnormality if the above hypothesis is correct.

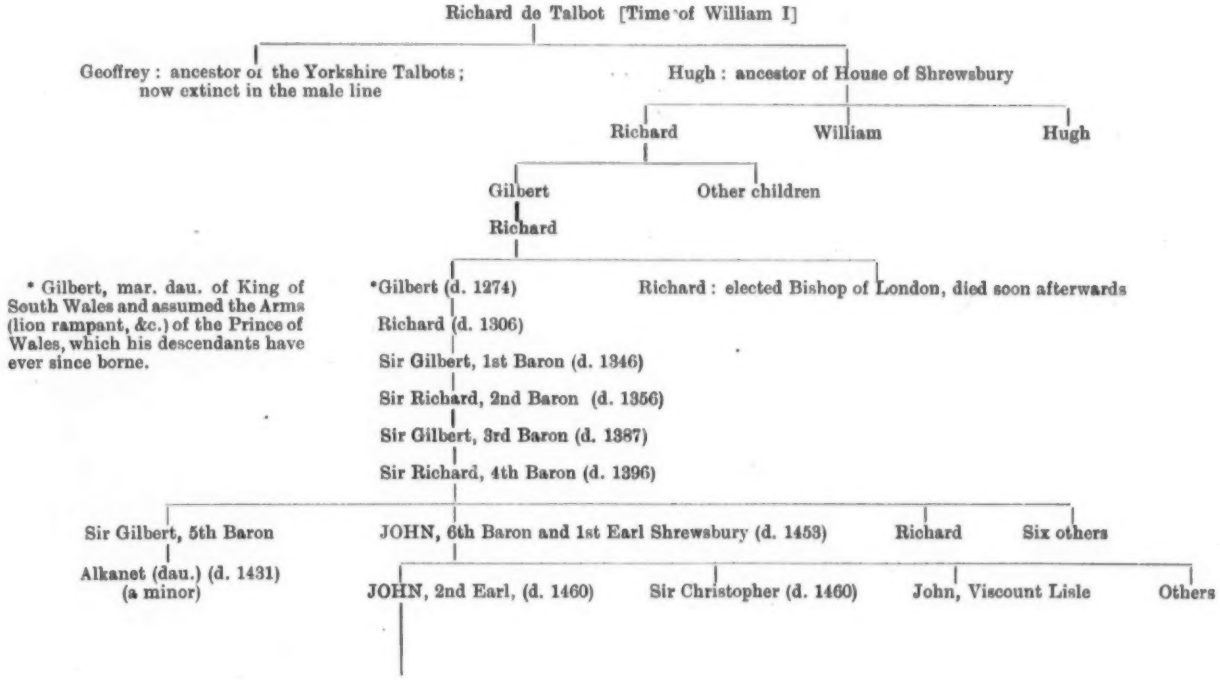
ADDENDUM.

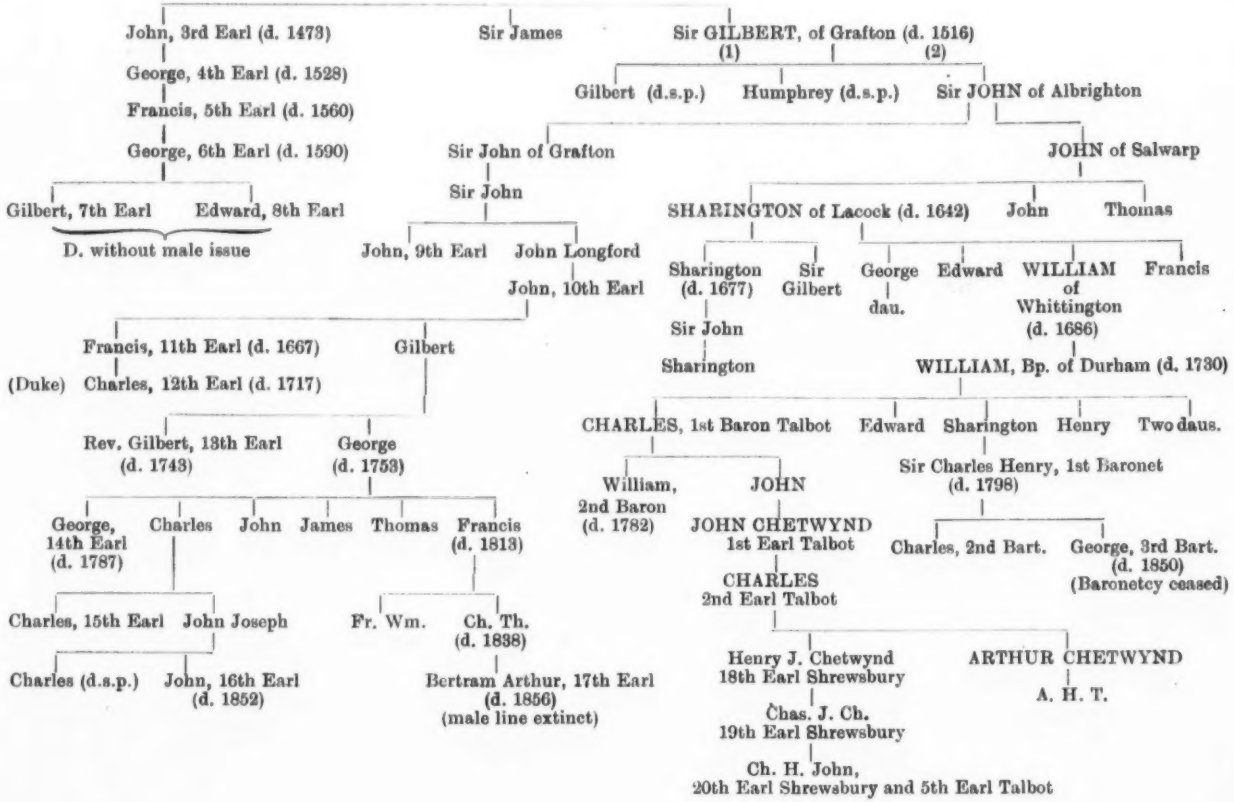
On April 24, 1916, I received from Professor Harvey Cushing, of Boston, U.S.A., a paper entitled "Hereditary Anchylosis of the Proximal Phalangeal Joints (Synphalangism)," reprinted from *Genetics* of January, 1916, and a letter asking me to try to find certain relatives of the family he described resident in this country. The condition described by him is essentially the same as that found in A. T., but I cannot trace any blood relationship between the two families. It is remarkable that when I received Professor Cushing's paper I was in correspondence with A. T., having heard of his abnormality on March 27, 1916.

Professor Cushing makes several references to my papers on Brachydactyly, but I do not think there is any genetic relationship between that condition and "Synphalangism." His paper is also illustrated with photographs showing the appearance of the hand when an attempt is made to flex the fingers. I regret not having secured a similar photograph.

What is the most appropriate name for this abnormal condition of the digits? If the term "ankylosis" is selected it is rather assuming too much, for, as Professor Shattock has reminded me, it is generally applied to a fixation of a joint, by fibrous or bony union, that has once been movable, and it is not known whether a movable joint ever existed in these fingers at the site of the missing articulation. "Anarthrosis"

A FAMILY PEDIGREE CONSTRUCTED FROM THE DATA GIVEN IN BURKE'S PEERAGE, SHOWING, IN CAPITAL LETTERS, THE MEMBERS WHO WOULD HAVE SHOWN THIS ABNORMALITY WERE IT TRANSMITTED AS A MENDELIAN DOMINANT.


Richard de Talbot [Time of William I]
Geoffrey: ancestor of the Yorkshire Talbots; now extinct in the male line
Hugh: ancestor of House of Shrewsbury
Richard
William
Hugh
Gilbert
Other children
Richard
* Gilbert, mar. dau. of King of South Wales and assumed the Arms (lion rampant, &c.) of the Prince of Wales, which his descendants have ever since borne.
*Gilbert (d. 1274)
Richard: elected Bishop of London, died soon afterwards
Richard (d. 1306)
Sir Gilbert, 1st Baron (d. 1346)
Sir Richard, 2nd Baron (d. 1356)
Sir Gilbert, 3rd Baron (d. 1387)
Sir Richard, 4th Baron (d. 1396)
Sir Gilbert, 5th Baron
JOHN, 6th Baron and 1st Earl Shrewsbury (d. 1453)
Richard
Six others
Alkanet (dau.) (d. 1431) (a minor)
JOHN, 2nd Earl, (d. 1460)
Sir Christopher (d. 1460)
John, Viscount Lisle
Others


John, 3rd Earl (d. 1473)
George, 4th Earl (d. 1528)
Francis, 5th Earl (d. 1560)
George, 6th Earl (d. 1590)
Gilbert, 7th Earl
Edward, 8th Earl
D. without male issue
Sir James
Sir John of Grafton
Sir John
John, 9th Earl
John Longford
John, 10th Earl
Francis, 11th Earl (d. 1667)
Gilbert
(Duke)
Charles, 12th Earl (d. 1717)
Rev. Gilbert, 13th Earl (d. 1743)
George (d. 1753)
George, 14th Earl (d. 1787)
Charles
John
James
Thomas
Francis (d. 1813)
Charles, 15th Earl
John Joseph
Fr. Wm.
Ch. Th. (d. 1838)
Charles (d.s.p.)
John, 16th Earl (d. 1852)
Bertram Arthur, 17th Earl (d. 1856) (male line extinct)
Sir GILBERT, of Grafton (d. 1516)
(1)
(2)
Gilbert (d.s.p.)
Humphrey (d.s.p.)
Sir JOHN of Albrighton
JOHN of Salwarp
SHARINGTON of Lacock (d. 1642)
John
Thomas
Sharington (d. 1677)
Sir Gilbert
George
dau.
Edward
WILLIAM of Whittington (d. 1686)
Francis
Sir John Sharington
WILLIAM, Bp. of Durham (d. 1730)
CHARLES, 1st Baron Talbot
Edward
Sharington
Henry
Two daus.
William, 2nd Baron (d. 1782)
JOHN
JOHN CHETWYND 1st Earl Talbot
Sir Charles Henry, 1st Baronet (d. 1798)
Charles, 2nd Bart.
George, 3rd Bart. (d. 1850) (Baronetcy ceased)
CHARLES 2nd Earl Talbot
Henry J. Chetwynd 18th Earl Shrewsbury
ARTHUR CHETWYND
A. H. T.
Chas. J. Ch. 19th Earl Shrewsbury
Ch. H. John, 20th Earl Shrewsbury and 5th Earl Talbot

seems to be free from objection, as it describes the present condition accurately. In the condition termed "brachydactyly," there is a condition of anarthrosis in the adult, but this is correctly termed ankylosis, because in every case there is in childhood free movement at all the joints, and the abnormal ones only become fixed and *obliterated* in later life, generally in early youth, as described in numerous instances in my papers on brachydactyly.

What has occurred in the family of A. T. cannot be known with any degree of certainty until one examines the radiograph of an affected child belonging to the family.

(*February* 13, 1917.)

Case of Congenital Hypertrophy of the Lower Limb.

By Hugh S. Stannus, M.D.Lond., Temp. Major, D.D.M.S. Nyasaland Field Force.

WITH A NOTE

By P. Perrow, Lieutenant S.A.A.M.C., Radiologist Nyasaland Field Force.

In two papers published in *Biometrika*[1] I have dealt with congenital anomalies met with in Nyasaland; further cases are noted in a paper by Dr. S. A. Kinnier Wilson and myself.[2] The following is published as an addition to the cases collected in those papers. It was met with casually during military operations against the southern part of German East Africa.

Kaliembe, of Mwamboneke village, New Langenburg District, German East Africa, male, aged about 17, of the Amwamba tribe, on examination presents a general hypertrophy of the right lower limb. His history makes it clear that this limb was larger than the other at birth and that the disproportion has persisted with growth.

[1] *Biometrika*, ix, Nos. 3 and 4, 1913; x, No. 1, 1914.

[2] "La Micromélie Humérale Bilatérale Congenitale," *Nouv. Iconog. de la Salpet.*, No. 6, November-December, 1911.

The father, mother, and elder brother and sister were all seen and examined, and presented no abnormality of growth, neither is there any history of such occurring in other members of the family. His father and brother are men of average height among their tribe, 172·5 cm. The mother is a small made woman with stature judged at 142·0 cm.

The subject of this article measured 154 cm. standing on his left foot. He was of poor physique, but except for the affection of the right leg and a similar, but minor change in the left foot, there were no physical signs of disease nor any other obvious anomaly of growth. There were no signs of acromegaly. The left foot presented an appearance of an overgrowth affecting the outer three-fifths of the part; while the hallux and second digit seemed normal in proportions, the outer three toes and the part of the foot behind them were enlarged, causing some distortion of the second toe by pressure, and some degree of varus.

Turning now to the right lower limb, little need be added to the information which the photographs and radiographs yield. These were very kindly taken for me by Lieutenant P. Perrow, S.A.A.M.C., Radiologist to the Nyasaland Field Force, to whom my best thanks are due. The deformity appears to be greater in degree as we pass from the distal to the peripheral part of the limb (figs. 1 and 2). The upper part of the thigh is little affected, while the buttock and parts entering into the hip-joint appear normal. Movement at this joint is normal. As we pass down the limb the increase in size is greater, so that the foot appears disproportionately larger than the rest of the limb, and judging by length alone might belong to a hypothetical man of about 6 ft. 10 in. in stature. The following measurements were taken:—

	Left leg	Right leg
(1) From anterior superior iliac spine to tubercle of fibula ...	44 cm.	51 cm.
(2) From tubercle of fibula to tip of external malleolus ...	40 ,,	50 ,,
(3) Total length of leg	84 ,,	101 ,,
(4) Circumference at middle of thigh	47 ,,	59 ,,
(5) ,, at knee	32 ,,	50 ,,
(6) ,, at calf	28 ,,	36 ,,
(7) Length of foot—tip of big toe to back of heel	26 ,,	35 ,,

The skin appears to unaided vision to be normal; there is no œdema. To touch the sensation yielded to the examining hand suggests abundance of loose subcutaneous fatty tissue; this is most notable about the knee-joint, which is very puffy looking. It is impossible

to distinguish the patellar tendon by palpation; and the skiagram suggested that it has probably stretched and atrophied.

The limb is carried in a position of partial eversion, the rotation being greatest at the knee, which is also partly flexed; movement at the knee is limited to about 5°.

FIG. 1.

Showing the position in which the hypertrophied lower limb is carried—viz., partial eversion—the rotation being greatest at the knee, which is also partly flexed.

I do not propose to enter here into the theories of the possible causation of such abnormalities; and owing to conditions of military service I am unable to give any references to the literature of the subject.

NOTE UPON THE RADIOGRAPHY OF THE LIMB BY MR. PERROW.

In studying the radiograms due allowance must be made for distortion inseparable from a limb of such enormous size. Bearing this fact in mind it may be observed that the femur is practically normal

FIG. 2.

An antero-posterior view of the hypertrophied lower limb.

in diameter but is certainly elongated. The patella is obviously misformed, and the head of the tibia deficiently developed. The portion of the tibia shown in radiogram I is abnormally large, whilst that of the fibula is extremely slender. The radiogram of the ankle shows a

normal articulation, but one of gigantic proportions. The fibula and tibia present their normal relations.

A radiogram of the right foot in a position of equinovarus shows what appears to be an articulation between the fourth and fifth metatarsals; apart from this and except for their unnatural size, the bones present a normal form and structure. The epiphyses of the four outer metatarsals and phalanges are ununited, while that of the os calcis is united—which would corroborate Dr. Stannus's estimate of the boy's age as being between 17 and 18. There is no extra epiphysis for the tuberosity of the fifth metatarsal, and no extra distal epiphysis for the first metatarsal.

A radiogram of the left foot demonstrates that the first and second toes are normal, whilst the third, fourth and fifth, with their coresponding metatarsals, are considerably enlarged, more especially in the long axis.

Section of Pathology.

President—Dr. F. W. Andrewes, F.R.S.

(*February* 13, 1917.)

Punctiform Hæmorrhages of the Brain in Gas Poisoning.

By F. W. Mott, M.D., LL.D., F.R.S., Major R.A.M.C.(T.).

The subject of punctiform hæmorrhages in the brain in gas poisoning has awakened new interest on account of its existence in fatal cases of shell shock with burial, and in cases of death from inhalation of poisonous gases, either as a result of their liberation by explosives in confined spaces, such as mines, trenches, and dug-outs, or as a result of an offensive gas attack by the enemy. I shall endeavour in this communication to explain the cause of these punctiform hæmorrhages.

In 1907 I published in the *Archives of Neurology and Psychiatry* (iii) a paper on "Carbon Monoxide and Nickel Carbonyl Poisoning." I came to the conclusion that the nickel carbonyl poisoning was really due to the inhalation of CO employed in the manufacture of nickel. Two such cases occurred of which I had the opportunity of examining the central nervous system, and I found multiple punctate hæmorrhages throughout the white matter of the brain, as you see in these photomicrographs.

In this paper I compared the naked-eye and microscopic appearance of the central nervous system in these cases of nickel carbonyl poisoning, with those observed in a case of suicide by illuminating gas, and I

considered them to be identical in nature. I also reviewed in this paper the clinical symptoms and pathology of CO poisoning, in respect to the findings in the central nervous system, and especially the causes which occasioned the hæmorrhages. All three cases died in from four to eight days with the complication of pneumonia. Thrombotic occlusion of the cerebral arterioles or venules was considered to be the cause of the hæmorrhages; in one case from the nickel carbonyl works an organized thrombus was found in a vessel of the medulla.

In the case of suicide by illuminating gas, admitted under my care at Charing Cross Hospital, signs of cerebral hæmorrhage occurred within twenty hours of commencement of the inhalation of the gas, for the limbs became rigid, and a plantar extensor reflex was obtained. At first the temperature on admission was 99° F., but when the rigidity of the limbs and the plantar extensor response was discovered six hours later, the temperature had risen to 105° F., and the pulse and respiration had become very rapid.

The nervous symptoms pointed to the occurrence of the punctiform hæmorrhages found post mortem in the internal capsules, and it may be assumed that the rise of temperature might have been due to the toxæmia coincident with the onset of pneumonia; for when the patient died, on the fourth day, pneumonic consolidation was found. Full notes of the clinical symptoms and post-mortem findings were reported. Microscopic investigation showed fatty degeneration of the heart and of the epithelium of the convoluted tubules of the kidney. Punctiform hæmorrhages, attributed to hyaline thrombosis of small vessels of the white matter, have been described by Bignami and Nazari in various diseases; for example, æstivo-autumnal malaria, apoplexy, diplococcal meningitis, following pneumonia and measles. It is possible, therefore, that pneumococcic toxæmia was productive of, or associated with, the causation of the hæmorrhages in these cases of CO poisoning.

But I am inclined to think that the CO poisoning alone would be capable of causing the punctiform hæmorrhages, for the following reasons: In both cases from the nickel works there was evidence of old hæmorrhages in the form of minute round or oval punctiform patches of softening, indicative of gas poisoning on some occasion previous to the man being obliged to give up work. And it was legitimate to attribute these symptoms they suffered with—namely, giddiness, vomiting, and

headache (migrainous attacks)—to the gas poisoning, causing congestive stasis and hæmorrhages. It is well to note that these migrainous attacks are frequently met with in men and officers who have been exposed to those conditions in which CO or other forms of gas poisoning might have occurred without fatal results.

From the facts observed in these three cases of CO poisoning, combined with certain anatomical conditions of the blood-vessels supplying the white matter of the brain, to which I shall now direct your attention, an explanation can be offered why these miliary hæmorrhages are found in the white matter of the cerebrum and basal ganglia, and not elsewhere in the brain. It must be recognized that a combination of factors may arise in CO poisoning—namely :—

(1) The heart, owing to the anoxæmia, has to beat faster, and to do more work with less oxygen; consequently it may undergo fatty degeneration.

(2) There is microscopic evidence of an irritative and degenerative endothelial change in the cerebral capillaries, as shown by mitosis of the nuclei, and a fatty degeneration made apparent by osmic acid staining. These changes may be due, as Lancereaux suggested, to CO in the serum, but aggravated by the pneumococcal toxin, which is also responsible for a tendency to increased fibrin formation of the blood, and to thrombosis in those vessels in which the anatomical conditions favour the lodgement of emboli, or clotting of the blood from congestive or inflammatory stasis.

The microscopic appearances of these punctiform hæmorrhages in known cases of CO poisoning, terminating in death by pneumonia, are similar to those shown in fig. 1 (p. 79), which is a section of the corpus callosum, showing punctiform hæmorrhages, from a case of shell shock with probable CO poisoning.

Miliary Hæmorrhages in Cases of Shell Concussion and Gas Poisoning.

I may now mention that the microscopic appearances found in these cases of CO poisoning, dying with pneumonia respectively after four days, eight days, and seven days, were in all respects similar to the appearances presented by sections of brains received from France, notified as dying of shell shock with burial and from gas poisoning—with one exception, and that only differed in the fact that

a large part of the hæmoglobin had been converted into chocolate-coloured pigment granules, which blocked the small vessels in the hæmorrhages.

Before proceeding to the description of these cases I will call attention to the anatomical relations of the vessels of the white matter of the cerebrum, where these hæmorrhages are found.

Anatomical Relations of the Vessels Favouring Capillary Stasis.

The pia mater covering the cortex sends delicate walled arteries and veins through the cortex to reach the subjacent white matter; the arteries consist of short and long vessels which, after giving off fine branches to the interlacing capillary network of the grey matter, terminate in a brush of fine arterioles; the short vessels end in this brush just below the cortex; the long penetrate deeper, to end in the corpus callosum and the centrum ovale. Each little artery breaks up into a tree, and forms a separate system of delicate arterioles. Each arteriole ends in a circumscribed area of capillaries, with an emerging vein. These veins do not anastomose. Thrombosis of arterioles or venules would therefore cause capillary stasis and hæmorrhage into the brain substance in a circumscribed area, also escape of blood into the perivascular sheaths of arterioles or venules; a condition generally found to occur where there are punctiform hæmorrhages. Owing to the thin character of the walls of the arteries, it is difficult to decide whether a vessel in section is an artery or a vein. Punctiform hæmorrhages are also found in great abundance in the brain structures supplied by the perforating arteries, especially those in which the opto-striate and lenticulo-striate branches terminate. These vessels give off relatively few branches until they reach their destination in the basal ganglia and internal and external capsules; they then terminate in a brush of delicate walled arterioles. Each vessel supplies, as in the case of the cortical vessels, circumscribed areas of capillaries, and the result of embolism or thrombosis is the causation of similar small limited areas of hæmorrhage and softening, which, when numerous, may become confluent. Stasis is favoured also by the narrow lumen of the capillaries of the brain.

Shell Shock and CO Poisoning.

The brain of a man said to have died from shell shock was handed to me by Professor Keith for examination. The following notes accompanied this brain :—

Fatal case of shell shock with burial, from Captain Armstrong, R.A.M.C., No. 7 Mobile Laboratory, B.E.F. Sent on from No. 1 Mobile Laboratory. No. 8 on Captain Armstrong's list.

Brain of man, admitted unconscious, with history of having been buried by shell blowing in parapet. Remained stertorous for two days and died.

Post-mortem Examination.—There is no wound of any kind on his body or head, and no visceral lesion. His ankle on one side was badly "sprained," but there were no fractures. The skull was unfractured, and no fracture of the base could be found. Brain shows multiple punctiform hæmorrhages and some slight subpial extravasation. No other particulars.

Photomicrographs illustrating the appearances presented by the brain in this case have been already published to illustrate my Lettsomian lectures to the Medical Society (1916).

Having regard to the fact that these punctiform hæmorrhages and hyaline thrombosis of vessels were identical in their microscopic appearances to those I had observed in CO poisoning, it occurred to me that the man may have been concussed and afterwards gassed while lying unconscious and buried (*vide* fig. 1).

It may be argued that these punctiform hæmorrhages were due solely to venous stasis and congestion, but I doubt this, for I have neither observed this condition in the number of cases of death from asphyxia occurring in status epilepticus nor after prolonged seizures of paralytic dementia, although I have examined the brains, macroscopically and microscopically, in a great number of instances.

A letter to me from the Trench War Committee confirmed the possibility of CO poisoning occurring when a large shell burst in a confined space, such as a dug-out or a trench, if incomplete detonation of the explosive occurred. Moreover, it must be remembered that CO is odourless, and may be present in trenches or dug-outs without its existence being known. When a mine is exploded considerable quantities of this gas may be formed, and it may travel through the ground considerable distances.

In the memorandum on "Gas Poisoning in Warfare" issued by the

Director-General, Medical Services, British Armies in France, it is stated in respect to CO poisoning—

> The lungs show no abnormal changes in cases of rapid death. Small punctate hæmorrhages may be found in the white matter of the brain, and sometimes ecchymosis in the meninges, if the case has been exposed to a concentration of CO sufficient to cause prolonged unconsciousness.

The fact that CO is not found in the blood when the patient is examined, does not prove that death was not due to CO poisoning, for after some hours of exposure to air it cannot be detected, and there is little opportunity for making the test for some hours or even days. Captain Dunn read a very interesting paper before the Medical Society on epidemic nephritis, in which he showed hyaline thrombosis of the vessels of the alveoli of the lungs and of the glomerular capillaries of the kidney.

In these cases he has observed multiple punctiform hœmorrhages of the brain, which he attributed to embolism by hyaline thrombi. These hæmorrhages present exactly the same appearances as in CO poisoning or gas poisoning. In a letter he has written to me, he states that he has now observed these hæmorrhages in four more cases of nephritis, so that their occurrence in the first case was not fortuitous. "They are of quite similar appearance to those I have observed in phosgene poisoning." He asks whether hæmorrhages of that type are seen in uræmia. Lieutenant-Colonel T. R. Elliott has forwarded me a memorandum by Captain H. W. Kaye on five autopsies on cases of poisoning by drift gas ($Cl_2 + COCl_2$) in which he describes blue-black dots in the brain of a seventy-hour case; he also refers to petechial hæmorrhages in the stomach and evidence of blood destruction in the spleen. Lieutenant-Colonel Elliott also calls attention to the fact that Captain Henry was the first to describe thrombi in the renal vessels and he disagreed with Dunn and McNee when they described renal emboli as coming from the lung.

Examination of the Brain in Gas Poisoning.

I have recently had the opportunity of examining the brains of two cases of gas poisoning, in which gas was employed in an offensive by the enemy; and one is of special interest.

The whole of the white matter is peppered over with small dark blue

spots about the size of a pin's head. These are due to hæmorrhages, but microscopic examination shows conditions which I have not found in CO poisoning, nor in other forms of gas poisoning; in fact, I have never seen any condition like this. The red blood corpuscles have been in large measure broken up, and the hæmoglobin converted into dark chocolate-coloured pigment granules, which fill the capillaries, arterioles and venules of the white matter of the brain. This is very possibly

FIG. 1.

Punctate hæmorrhages in corpus callosum from a case of shell shock and burial; very probably accompanied by gas poisoning while lying unconscious and buried. Observe the small white area in the centre of the hæmorrhage, in the middle of which is a small vessel which, under a higher magnification, will be seen to contain a hyaline thrombus. (× 20)

methæmoglobin, for it is known that exposure to nitrous fumes in concentration will oxidize the hæmoglobin, and convert it into methæmoglobin. Phosgene, $COCl_2$, has been used by the Germans

it liberates HCl when it comes in contact with a moist surface; it is very irritating and would cause bronchiolitis, and I am informed by my friend Professor Halliburton that it is possible the free hydrochloric acid would convert the hæmoglobin into acid hæmatin.

Similar appearances were found to those described in CO poisoning—namely, multiple punctiform hæmorrhages in the white matter—but the blood corpuscles were intermixed with chocolate-coloured pigment granules (Plate I). The accompanying drawings illustrate the appearances presented. You may observe a vessel with a hyaline thrombus, stained pink by the fuchsin of the Van Gieson stain, but

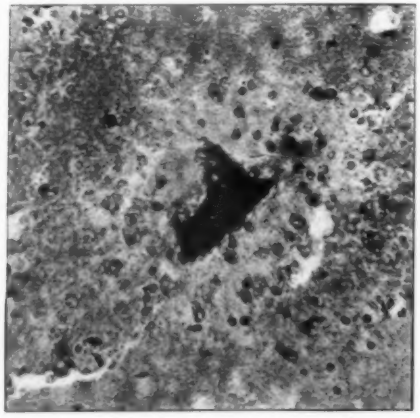


FIG. 2.

Hyaline thrombus of vessel in centre of a punctate hæmorrhage. The thrombus was stained brown by dissolved pigment. Around the blocked vessel is a white area of brain substance containing numbers of leucocytes; outside this is the hæmorrhage, not very distinctly seen. The specimen was prepared from the subcortical white matter of the frontal lobe. (× 345.)

with a brownish tinge due to the change in the blood pigment (Plate II).

Possibly, as in capillary fat embolism, we may have embolism by these pigment granules, but, generally speaking, there is definite evidence of thrombus formation, with pigment granules in the thrombus.

PLATE 1.

Section of optic thalamus, showing vessel blocked with pigment going to hæmorrhage; amidst the blood corpuscles are numerous pigment granules. To the left of the larger vessel are three capillaries packed with pigment and compressed together. (× 350.)

MOTT:
Punctiform Hæmorrhages of the Brain in Gas Poisoning.

PLATE II.

Small vessels blocked with pigment in hæmorrhage, and to the right a larger vessel, probably a vein, filled with lightly brown-stained hyaline thrombus. (× 150.)

Examination with a high-power magnification shows that the smallest vessels are filled with the pigment, which can be seen in the vessels and in the circumscribed areas of hæmorrhage as discrete granules packed together (Plates I and II).

The specimens show hyaline thrombus formation in the vessel contained in the centre of the hæmorrhage or passing to the centre of the hæmorrhage; the walls of these vessels are generally so thin as to support the view that they are veins (fig. 2). Some of the small vessels

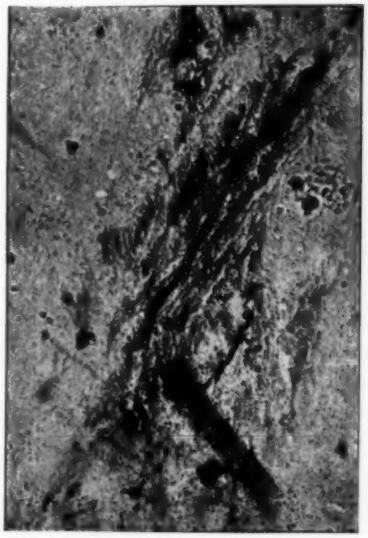


FIG. 3.

Small vessel breaking up into a leash of small arterioles compressed together by the hæmorrhage. The vessels are all blocked with black pigment granules. Specimen from internal capsule gas poisoning. (× 70.)

show aneurysmal dilatation. In one section I observed a leash of small arterioles pressed together by the hæmorrhage at the side; on one there was an aneurysm filled with pink-stained thrombus (figs. 3 and 4). Amidst the corpuscles are numbers of pigment granules. Three such hæmorrhages with occluded vessels proceeding to them can be seen (fig. 5, p. 83). Nearly all the punctiform hæmorrhages show a central

vessel, surrounded by an area of necrosed brain tissue, infiltrated usually with leucocytes (fig. 2, p. 80). The whole of this area is stained pink by Van Gieson's stain, and it is more or less difficult to make out the wall of the central vessel. Sometimes a capillary filled with a thrombus can be seen running to the central vessel. It may be filled with chocolate-coloured pigment granules probably embedded in a coagulum as in Plate I, or the coagulum may be of a pinkish-brown colour due to the

FIG. 4.

Leash of small perforating optostriate arteries filled with pigment granules. Two of the arterioles show miliary aneurysms. (× 350.)

coagulum being stained by the pigment dissolved in the serum (Plate II). In this, as in all other cases, there is evidence of an inflammatory stasis and excess of leucocytes in the vessels, and often into the perivascular sheath and tissues around.

Clinical Anatomical Notes accompanying this Case.

Brain.—Surface veins large and small, distended with dark blue clotted blood (veins of base of skull were in same condition). Section shows thickly scattered blue-black dots throughout the brain, especially in the white matter; this applies also to the cerebellum, and to much less extent to the pons and medulla.

No hæmorrhage seen.

Patient was admitted ten hours after being gassed, and died sixty hours after admission, from bronchiolitis and failure of right heart.

FIG. 5.

Three punctate hæmorrhages showing optostriate arterioles filled with pigment granules. (× 30.)

It is unfortunate that the clinical and post-mortem notes of this case are so scanty, for it is one of great pathological interest. The right heart failure and bronchiolitis, from which the patient died seventy hours after inhalation of the gas, would undoubtedly account for the venous congestion and stasis noted post mortem, and for the thrombosis of the small vessels in the white matter of the brain. The blocking

of the capillaries, small arteries, and veins by the chocolate-coloured granules of pigment, especially of the capillaries, would, however, suffice to account for the hæmorrhages. In some respects the capillary blockage by pigment resembles the condition found in pernicious malaria, in which disease Bignami and Nazari have described punctate hæmorrhage of the white matter of the brain; but I am inclined to believe the principal cause of the hæmorrhages is inflammatory stasis and hyaline thrombosis of arterioles, capillaries, and venules, the pigment granules being incorporated in the coagulum.

I received the brain of another case in which the bruises on the body, the hæmatomata in the right lung, and the other conditions described, all suggest that he had been blown up by a shell and buried, and that the injuries of the brain were due to concussion. The fact that there was no CO detected in the blood does not conclusively prove that he was not exposed, while buried, to CO gas.

Clinical Notes of this Case.

Admitted with diagnosis of shell shock. Purple bruises on arm and leg of right side. Stertorous, unconscious, and during the night before death, constant fits. Lived thirty hours in hospital.

Post Mortem.—There were two hæmatomata in the right lung, but no other visceral injury. No hæmorrhage of the scalp, and no fractures of the skull. Some slight subpial hæmorrhage of the right hemisphere. Fornix destroyed and full of hæmorrhages; hæmorrhages also seen in corpus callosum. Hæmorrhage in both optic thalami; cerebrospinal fluid tinged with blood. Men admitted with him said he had been buried by a shell. There was no CO in his blood, and the bruising was purple.

Microscopic Examination.—Multiple punctate hæmorrhages are seen; hyaline thrombosis of capillaries, arteries, and venules; perivascular sheaths contain blood (Plate III). Marked evidence of inflammatory stasis. Some of the small veins are filled with blood corpuscles, one-half of which are polymorphonuclear leucocytes, and in the perivascular sheath and tissues around are large numbers of polymorph leucocytes.

PLATE III.

Section of frontal cortex from case of shell gas poisoning. Hyaline thrombus of vessel in the centre of hæmorrhage. (× 150.)

W. O. C. 862.

REPORT ON A CASE OF "SHELL GAS POISONING," ADMITTED INTO 141 SECUNDERABAD CAVALRY FIELD AMBULANCE ON AUGUST 31, 1916, WHICH TERMINATED FATALLY ON SEPTEMBER 5, 1916.

No. 1489 S. D. had been one of a working party which was caught in a "gas-shell barrage" put up by the enemy on the morning of August 31, at about 10.30 a.m. The majority of the shells on bursting made a small explosion and formed a quickly dissipated white smoke. At the time he was standing in a trench 3 to 5 ft. deep, and appeared to have been knocked down by the explosion of this particular shell. Whether or not he had on an anti-gas helmet at the time is uncertain, but it appears that after the man next to him was wounded they both removed their anti-gas helmets, and were found lying in the bottom of the trench. He was soon placed on a stretcher and removed to a dressing station. As to whether he received any definite medical treatment prior to his admission into 141 Cavalry Field Ambulance at 7 p.m. is uncertain.

On admission: Patient was unconscious and frothing slightly at the mouth and nostrils. He was distinctly collapsed. There were no signs of cyanosis. (*a*) Pulse 112, regular, but weak. (*b*) Respirations 53, deep, no signs of obstruction. (*c*) Temperature 96° F. Patient was very restless and had a slight cough. There was a small superficial scalp wound behind his right ear, and also a superficial wound with considerable bruising of tissues round about on back of left shoulder.

Physical signs: A few râles could be heard over the front of the chest, but nothing abnormal at the back. There was no evidence of accumulation of fluid.

Treatment: Immediately on admission $\frac{1}{60}$ gr. strychnine was given hypodermically. Ammonia capsules were administered from time to time. Oxygen was administered (through the mask of a box respirator) for four minutes every quarter of an hour. The oxygen for a time quietened the breathing, and the frothing at the mouth and nose ceased.

10 p.m.: Pulse 120, weak and irregular. Respirations 40, deep, and no obstruction. Temperature 96·4° F. Patient was again given $\frac{1}{60}$ gr. strychnine.

11 p.m.: Ammonia capsules were ordered to be used continuously. At first there was considerable improvement in the pulse, although the rate remained the same (120), but this improvement was only temporary, and at 11.30 p.m. the pulse again became very feeble and irregular and its rate now was 148.

12 p.m.: No signs of improvement. Patient was unable to swallow.

3.15 a.m.: Patient was losing ground rapidly. The pulse was almost impossible to count. Respiration 60. Temperature 97° F. Patient was very restless.

Oxygen had been administered continuously for four minutes every quarter of an hour since admission. The administration of same seemed to bring on the restlessness of the patient, and as there was no evidence of cyanosis and no respiratory obstruction the oxygen was stopped. $\frac{1}{60}$ gr. strychnine was again given.

6 a.m.: Evidence of slight improvement, and patient was able to swallow a little milk. He also showed signs of returning consciousness. Pulse 120, more regular and fuller. Respiration 50. Temperature 97° F.

8 a.m.: Still more signs of improvement and more conscious. Was able to take more milk. Pulse 100. Respiration 50. Temperature 98° F. $\frac{1}{60}$ gr. strychnine again given, and also $\frac{1}{30}$ gr. digitalin.

11 a.m.: Patient was still very restless, but was more conscious, and was able to take a little chicken extract. Pulse 84, full and regular. Respirations 60. Temperature 97·2° F. Condition generally was one of improvement. Patient passed a motion of which he was quite unconscious. He was given 1 c.c. pituitary extract hypodermically, and this treatment was continued four-hourly with intermittent doses of digitalin or strychnine. Numerous râles could now be heard all over the chest, both in front and behind, but there was no sign of dullness.

3.30 p.m.: Condition was much the same. Still very restless and slightly less conscious again. Passed another motion. Although patient was conscious of passing his motions, he passed his urine quite unconsciously.

5 p.m.: Patient was slightly worse again and distinctly less conscious. Pulse 128, less full and more irregular again too. Respirations 58. Temperature 100·2° F. 1 c.c. pituitary extract again given hypodermically. Ammonia capsules were now being used at the rate of one every half hour.

8 p.m.: Condition much same as at 5 p.m.

11 p.m.: Showed signs of slight improvement. More conscious and less restless. Pulse 92, regular. Respirations 46. Temperature 99° F.

September 2, 2 a.m.: Condition much same as at 11 p.m. on September 1. Pulse 102. Respirations 50. Temperature 98·2° F.

6 a.m.: More signs of improvement. Patient was more conscious, and was only restless at intervals. Passed another motion. 1 c.c. pituitary extract was again given, and this was the last dose administered for some days. Pulse 84, regular and full. Respirations 36. Temperature 97·8° F. Hot water bottles were applied.

10 a.m.: Condition much same as at 6 a.m., but patient was slightly more restless again. Numerous râles could still be heard all over the chest, both in front and behind, but again there was no evidence of dullness or fluid accumulation.

4 p.m: Patient's condition very satisfactory. He had slept from 3 p.m. Pulse 98. Respirations 40. Temperature 99·4° F.

8 p.m.: Progressing favourably. Slept for fifteen to twenty minutes at frequent intervals. Pulse 95. Respirations 40. Temperature 99·6° F.

Ordered ammon. carb. 5 gr. t.d.s. The patient was now able to take milk and chicken extract with ease and in appreciable quantities.

From 8 p.m. on September 2 till 9.30 a.m. on September 5: Patient showed signs of great improvement. He slept well, sometimes for two to three hours at a stretch. He was able to take milk, chicken extract, sago and soups. His bowels moved daily, and although he was quite conscious of passing his motions, he still never was conscious of passing his uriue. During the period the only medicine prescribed was ammon. carb., 5 gr. t.d.s. Pulse varied from 95 to 62, was always regular and strong. Respiration rate gradually decreased from 40 to 28 per minute. Each respiration was always full and deep and there never was any sign of respiratory obstruction. Numerous râles were audible, both in front and at back of the chest. No dullness was ever detected. Temperature gradually came down from 99·6 to normal.

For the first thirty-six hours after admission the patient was kept lying on a stretcher in the open, but after that he was placed in a well ventilated room.

10 a.m.: At 10 a.m. on September 5, the patient suddenly showed signs of getting worse again. His breathing became more rapid, and to a certain extent became laboured too. His temperature fell from normal to 97° F. His pulse became rapid and weak, and he showed rapidly increasing signs of delirium. Given $\frac{1}{60}$ gr. of strychnine hypodermically.

2 p.m. to 6 p.m.: Patient was rapidly losing ground. Quite unconscious; vomited once. Pulse-rate rose from 84 to 146, very feeble and irregular. Respiration: Breathing became stertorous and shallow, and mainly abdominal in type. The rate of breathing first increased from 32 to 40 (at 2.30 p.m.) and then gradually decreased to 16 per minute (at 6 p.m.). Temperature at 2 p.m. was 96·4° F.; at 3 p.m., 96·4° F., and then again rose to 98·6° F. at 6 p.m. Patient was packed round with hot water bottles. He was given another $\frac{1}{60}$ gr. strychnine and shortly afterwards 20 minims of spirit. ammon.

6.30 p.m.: Condition very grave. Breathing very stertorous in type and very shallow. Rate of breathing 20 per minute. Pulse 140, very feeble and irregular. Temperature 99° F. Patient by now had developed very marked nystagmus, internal strabismus, and his right pupil was distinctly sluggish and slightly larger than the left.

7 p.m.: Condition still worse. Pulse 126, very feeble. Temperature, 100·2° F. Respiration very stertorous.

7.38 p.m.: Patient died.

Post-mortem Observations.—A post-mortem examination of the brain was made on September 6, and the following noted: No injury to the cranium, either externally or internally, at the site of the scalp wound. No evidence of hæmorrhage either on the surface of the brain, or at the base of the skull.

On Section.—(1) Cerebrum: Numerous punctate hæmorrhages scattered throughout both the grey and white matter, and in the grey matter of the right lobe there was one hæmorrhage about the size of a split pea.

(2) Cerebellum: Numerous punctate hæmorrhages in cerebrum, but in

addition there was one large hæmorrhage about the size of a shilling on the right side, and one about the size of a threepenny-piece on the left side.

(3) Pons: Scattered throughout its substance too, these small punctate hæmorrhages were noticed.

(4) Medulla: Same as the pons.

(5) Fourth ventricle: Both the roof and the floor of the fourth ventricle were studded with numerous punctate hæmorrhages which here were so numerous as to appear almost like one continuous large hæmorrhage.

(Signed) J. W. VAN REENERD, Lieutenant, I.M.S.

The following points are, I think, of interest in connexion with the gas employed in this case, the composition of which I do not know.

(1) It was one that had two different toxic actions—viz., primarily on the cardiac centres, and secondly, much later, on the vascular system, causing a degeneration of the walls of the small vessels, leading to hæmorrhages into the substance of the brain, and probably other organs as well, but they were not examined. There were no serious symptoms apparent at any time of toxic actions on the respiratory centres or organs.

(2) The late appearance of the fatal symptoms, or perhaps it would be more correct to say the delayed action on the vascular system. The patient had survived the critical period of cardiac failure, and was apparently well on his way to recovery when the fatal symptoms appeared—five days after he had been gassed.

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Unfortunately there are no notes of the condition of the reflexes nor the state of tonus of the muscle of the limbs. The clinical notes do not indicate that this patient suffered with pneumonia, nor any obstruction to the entrance of air to the lungs; there is no statement regarding the cause of the extremely rapid respiration, but the fact that he was given oxygen and diffusible stimulant for the first twelve hours suggests that there was great respiratory embarrassment due to deoxygenation of blood. Later there is a definite statement; there is no evidence of cyanosis and no respiratory obstruction; the oxygen was stopped. But the respiration still continued very rapid—50 to 60. After some days his condition greatly improved, and the respiration fell even to 28. Then on the last day in the morning he suddenly developed grave symptoms, and in the evening it is noted that he developed marked nystagmus, internal

strabismus, and the right pupil was distinctly sluggish and slightly larger than the left. The conclusions and findings are not inconsistent with CO poisoning, but it may have been some other gas or mixture of poisonous gases.

I was unable to confirm the statement of hæmorrhage into the pons and medulla. The vessels were congested, but no hæmorrhage was found. The cerebral hemispheres were badly preserved, and I was only able to examine the cerebral cortex of the frontal lobes.

Microscopic Examination.—Portions of the frontal cortex and subjacent white matter, showing to the naked eye miliary punctiform hæmorrhages, were taken, and, as in all the other cases, blocked in paraffin, and sections cut and stained by Van Gieson and hæmatoxylin-eosin methods, also with polychrome. The punctiform hæmorrhages appeared in some of the sections to form a circle of circumscribed, discrete, oval or round areas of extravasated blood, often with a section of a vessel in the centre or proceeding to the hæmorrhagic area. In sections stained by the Van Gieson stain the lumen and the thin-walled vessels so seen appear a pale pink, and this presumably is due to the contained hyaline thrombus (Plate III).

In some vessels red blood corpuscles are seen with abundant fibrin formation; a similar appearance to that seen in the alveoli in red hepatization; other vessels appear filled with polymorphonuclears and fibrin. Around the central thrombosed vessels of the hæmorrhage are seen deeply stained pink areas of necrosed brain tissue infiltrated with polymorphonuclear leucocytes; very often a vessel can be seen filled with blood which is extravasated into the sheath, and occasionally the rupture of a thin-walled vessel causing hæmorrhage into the perivascular sheath can be seen. This condition of central thrombosis with necrosis of brain tissue around and infiltration of leucocytes is similar to that observed in the CO poisoning from the nickel works, when the patient lived eight days; and is in accordance with what might be expected, seeing that the man lived six days after possible inhalation of the gas.

Summary.

The reason why these punctiform hæmorrhages occur in the white matter of the brain is primarily due to the anatomical condition of the vessels in the white matter of the cerebrum, where the arteries are terminal; each small artery having a separate capillary system, likewise the emerging veins. A tendency to stasis may be brought about in

these separate vascular systems by the failure of the heart as a force pump and suction pump, also by those respiratory conditions which lead to right heart dilatation, and interference with the return of blood from the skull. In most cases the two factors are combined. It seems probable, however, that either factor may act independently as a coefficient of gas poisoning in causing inflammatory stasis and thrombosis, resulting in multiple punctiform hæmorrhages. In the gas case, in which the hæmoglobin has been converted into pigment granules, it seems probable that the hæmorrhage may be accounted for by embolic occlusion of the arteries. It is unfortunate that with the exception of the case of CO poisoning by illuminating gas I have not had the opportunity of examining the organs of the body.

It is quite probable that, as in that case, fatty degeneration of the heart, the kidneys, liver, and vessels of the brain would be found to exist.

In conclusion, I desire to acknowledge my indebtedness to my assistant, Mr. Charles Geary; also to Miss Munro and Miss Watson for the assistance they have afforded me, and to the Board of Control for a grant to enable me to give them an honorarium for their services.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

J. Y. W. MacALISTER

UNDER THE DIRECTION OF

THE EDITORIAL COMMITTEE

VOLUME THE TENTH

SESSION 1916-17

SECTION OF PSYCHIATRY

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1917

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LONDON:
JOHN BALE, SONS AND DANIELSSON, LTD.,
OXFORD HOUSE,
83-91, GREAT TITCHFIELD STREET, OXFORD STREET, W. 1.

Section of Psychiatry.

President — Dr. R. Percy Smith.

(*October* 31, 1916.)

PRESIDENTIAL ADDRESS.

Mental Disorders in Civilians arising in Connexion with the War.

By R. Percy Smith, M.D.

My first duty is to express my thanks to the Section of Psychiatry of the Royal Society of Medicine for the honour it has conferred upon me in electing me as its President. The Section is still one of the youngest of the Sections of this Society, and as its third President I feel that it is a special honour to succeed men of such reputation and power of original thought and research as Sir George Savage and Major F. W. Mott, and I trust that the members of the Section will give me their indulgence if I fail to reach the high standard which has been set by my predecessors.

In the past two years of the stress of war, when a large part of one's daily time has been taken up by reading the newspapers with news from the Front, when one's emotions have been stirred by reading of deeds of heroism and self-denial on the one hand, and of the distresses of the population in invaded countries, cruelties to wounded prisoners, callous indifference and neglect of the sick, as at the Wittenberg Camp, and attempts at terrorizing the people of this and other countries by various methods of "frightfulness" on the other, it has been difficult to concentrate one's attention on any scientific work.

In this respect I speak only of my own mental state, as the Section is of course well aware of the valuable contributions made by Major Mott, Major Rows, and others, towards the clinical and psychological problems presented by the large number of soldiers returned from the

Front, suffering in various ways from mental or nervous disorder arising from the intensity and prolonged strain of modern warfare.

Much has been written both in British and foreign medical journals with respect to mental and nervous disorders occurring in soldiers as the result, either entirely or partially, of the stress of war. I have, however, been able to find little reference in recent literature to the effect of the War in the production of morbid mental states among the civilian population, and it seemed to me desirable to put on record my experience as to this, and to inquire whether any and what cases can be shown to have had mental disorder produced wholly or partly by the War, and also what has been the effect of the War on pre-existing psychoses, and as far as can be at present ascertained on the increase or otherwise of insanity in the British Isles.

I am therefore excluding from this paper all the cases of soldiers who have broken down mentally after suffering the strains and horrors of trench warfare and shell shock and severe wounds, although I shall refer to some of those who, following civilian occupations before the War, have enlisted voluntarily or been given commissions and have broken down mentally either before they have actually been abroad or before they have seen any fighting at the Front. My chief reference will be to cases occurring in civil practice and chiefly those seen in consulting work.

It may be well to turn back to the period immediately preceding the War. In July, 1914, came the news of the assassination of the Austrian Archduke and his wife at Serajevo with the rapid development of threats by Austria to Serbia, the subsequent intervention of Russia, Germany and France, the invasion of Belgium by the Germans and the resulting participation of this country in the world-war. It may be safely said that a month before the declaration of war on August 4, 1914, the general population in these islands had no conception whatever that in a short time we should be involved in the greatest war in history and up to the very moment of the declaration there was a considerable party both in and out of Government circles who were utterly opposed to the participation of this country in it. Underlying the desire for peace and for avoiding the horrors of war, there lay however the "complex" involved in the idea of the sacredness of England's word as expressed in a treaty, and in by far the largest section of the population there was a sense of relief at the fateful decision.

On the Sunday evening before the declaration of war I was in Trafalgar Square, where thousands were gathered on the outlook for

news, and in the expectation of a fateful decision one way or the other. Apparently a "herd-instinct" had taken possession of numbers present as very shortly it became evident that crowds were, without any direction from any leader, moving along the straight road to Buckingham Palace. There was no noise nor excitement, but just a steady march of men and women as if all were swayed by some common instinctive impulse to move towards the head of the nation. Arrived in front of the Palace the crowd stood for a time almost voiceless, but emotion could not be suppressed and very soon was expressed by repeated cheering and singing of the National Anthem, eventually gratified by the appearance of the King and Queen on the balcony, whereupon after a prolonged outburst of enthusiasm the crowd silently dispersed, having given this expression of its affection and support to the head of the nation.

There is no doubt that since then the emotions of the people of this country have been constantly and deeply stirred, though, as is our characteristic, they are not always actively expressed. A nation is, however, made up of individuals, and we who are engaged in the practice of psychiatry are thoroughly familiar with the frequency with which emotional disturbance, particularly painful emotion, precedes the onset of insanity. Therefore it is not at all surprising that the stirring of emotions associated with the greatest war in the world's history should appear to be responsible for a certain amount of insanity in the civilian population. Although the chief stress of battle lies on the soldiers at the Front, those at home suffer in another way. After the first wave of enthusiasm at the nation having come to a right decision, or at any rate a decision involving a most strenuous line of conduct to be carried out to a conclusion of defeat or victory, comes what may be called the "cold fit" of counting the cost, the parting from fathers, brothers or sons, perhaps never to return, or perhaps to return damaged irretrievably in health or limb, the news that they are or have been in action, the watching of lists of killed, wounded, missing, or prisoners, anxieties about civilians interned abroad, and the thousand and one ways in which anxieties about those at the Front affect those at home.

The onset of the War had a particularly disturbing effect on those who were on the verge of breaking down or had had previous attacks of mental disorder. I give here some of the cases I have seen:—

Case I.—B., aged 38, architect, had had a previous attack of insanity in 1905, and was ill for about a year but had since resumed work. In July, 1914, he had passed an examination lasting four days, and had been working very hard. On August 12, 1914, he had become excited about the War and

was sleeping badly, and constantly thinking about its prospects. He complained that he did not feel well, then quickly passed into a state of excitement, was rushing up and down, talked about the Germans, said he would show them "what was what," quickly became confused, saw imaginary daggers and bullets and talked to imaginary people; talked constantly about the War, said he was really Napoleon, shouted out "Damn the Kaiser," and wanted to go out of the window and fight the world. In a short time the excitement passed off and he appeared to recover, but quickly relapsed when at liberty, and had to be placed under care again with recurrent attacks of excitement, and is still under care.

Case II.—Miss W., County School teacher, aged 20. Mother neurotic, but no definite insanity in family. Had an attack of tonsillitis in August, 1914, which laid her up for a fortnight; was very weak and run down afterwards. While recovering, her fiancé enlisted, much against her wish. Began to be very worried and anxious about this, slept very badly, and said she heard him walking up and down outside the house. The War, and anything bearing on it, then wove itself into her ideas. She said that a Captain whom she had met about a year and a half before, while on a summer holiday, and who was already at the Front, was a spy and had an assumed name; thought he had put diphtheria germs into the water; that the spy had found out all about her fiancé and had hypnotized her; said she had killed the Kaiser because God conquers materialism, and also that she had overcome the spy and won a moral victory. This acute confusional attack necessitated treatment in a mental hospital, where she recovered.

Case III.—Miss P., aged 22. Maternal grandfather epileptic and insane, maternal aunt insane, sister insane. For about three months before the War began she had been somewhat suspicious, thought people said she had had a child by a man from whom she had riding lessons and for whom she had formed an attachment. The onset of the War seemed to change the current of her ideas. She said we had got into a new world; that the questions about Germany, Austria, and Belgium had been in 1914, but that now the end of the world had come; that the Kaiser was Antichrist; called the cat the Kaiser, and the kitten Belgium. She passed through an episodic state of confusion and delirium, which passed off, leaving the earlier ideas again prominent for a time, though they eventually disappeared.

Case IV.—Mrs. D., aged 57. Father insane, brother phthisis. She had been twice under care at Bethlem Hospital in 1898 and 1908, recovering each time. She was quite well till the War began, then read the daily papers a great deal; became depressed and worried; thought her husband had a double personality, and that he would give her into the power of the Germans or was going to kill her; was afraid of "German looking" people; had doubts and fears of people in the street; thought her husband was in league with evil spirits or was perhaps Mephistopheles.

Case V.—Miss J., aged 43. Father had hypochondriacal melancholia; sister depressed; mother died of pernicious anæmia. For some months before

the War she had been concerned about the state of her bowels, but attributed a more acute condition, which subsequently developed, to what she called "that fright"—that is, the outbreak of the War. She said that, from that moment, "everything had gone rigid inside," everything was stuck up, and that both the front and back passages were "dry"; that the contents of her bowel had accumulated to such an extent that there was nowhere to put it. She made considerable improvement for a time, but the prognosis for the future was unfavourable.

Good examples of the effect of extreme anxiety about relatives are the following :—

Case VI.—A married woman, aged 47, had been somewhat addicted to alcohol, and was at the climacteric; no inheritance known. Her son was taken prisoner by the Germans while serving in the R.A.M.C., and was threatened to be shot as a spy. She at once began to be depressed and to take alcohol; became fearful, and dreaded losing self-control; had impulses to strangle people, and tried to choke a nurse; felt impelled to kill her husband, and to strip off her clothes and put herself in the fire; begged to be put in safety; said she ought to be tied up, and that it would be right to kill a lunatic. The symptoms abated to a certain extent after the release of her son, but she had frequent relapses, and is still under care.

Case VII.—Mrs. S., aged 66. Paternal aunt insane, brother and sister odd, daughter depressed. She had always been neurotic. Her son was in the Army and came home on leave; she was then apparently well, but immediately after his return to the Front she collapsed, became depressed, and agitated; saw ruin and fire, and said she heard "rain of fire" in the night; dared not look out of the window; thought that the house would be on fire and there would be no water to put it out; that her food was poisoned, and her bowels were blocked up; that she was wicked, and they were all going to hell. She had to be placed under care.

Case VIII.—Mrs. B., aged 36. No family history. Probably tendency to alcohol. Husband was in the merchant service engaged in transport work. She went to America with him in his ship at the beginning of the War and stayed there while he was on voyages; was in constant fear of danger to her husband from submarines; thought everyone was pro-German, and gave way to violent outbursts against the Germans; then began to think she heard voices chanting about her and aspersing her character, and became suicidal. After being in a sanatorium in the United States was brought to England; constantly heard voices while on the voyage saying she was to be divorced, and thought she was insulted through "gramophones." She had to be placed under care on arrival.

Case IX.—Mrs. R., aged 54. Brother committed suicide. She had had a previous attack of depression twenty years before; she had lost her husband in 1914. Her son was killed at Gallipoli; was much upset by this. His widow lived with her doing war work. She began to sleep badly and took

paraldehyde secretly for it, then became depressed, silent, and monosyllabic, thought spirits talked to her, and passed into an attack of melancholia.

Next to stress and anxiety about those at the Front, or the shock of their death, come the "res angustæ domi." Especially in this War, where not only the professional soldier has been called upon to carry out his duty, but where armies of a size totally unprecedented in this country have had to be raised; where the bread-winner has had to relinquish his comfortable peaceful occupation and his home and do arduous and dangerous duties at a remuneration at which he would have scoffed in peace time; where, if, for example, he has been a member of the medical profession, he has had, as in many cases, to relinquish a good practice, to give up his house, to take away his children from school with the very grave doubt as to whether he will ever be able to reinstate himself if he returns to civilian life, very considerable trials, stresses, and anxieties have fallen upon the wives and families.

It is true that in many cases the "separation allowance" has provided an amount of money that had never been seen in the household before, but there are sections of the population, perhaps chiefly the professional classes or those with small fixed incomes, where the making of the ends to meet has been a constant worry, and, as is well known, adverse circumstances and pecuniary difficulties have long been regarded as contributory causes to insanity. To many the increased cost of living and diminished income has been one of the causes leading to mental illness. Examples of this are the following:—

Case X.—Mrs. M., aged 57. Great aunt insane, sister weak-minded. Separated from husband twenty-five years but he lived in the same town. Catamenia ceased five years. Was much upset by the outbreak of the War in August, 1914; became depressed and sleepless; said she must not spend money; thought she was a "sneak" to change her house while the War was on; talked about the War upsetting her sleep; developed the delusion that she was not able to pay for anything and would be locked up in consequence; thought she had brought ruin to the house, and damned everybody; thought cocks crowed at her, and that everyone considered her a bad woman. She had to be placed under care.

Case XI.—Mrs. C., aged 40. Grandmother had climacteric insanity. Had had a very unhappy married life and had divorced her husband in April, 1914. Four years before had had delusions, but recovered. In October, 1914, had operation for appendicitis and recovered. Then was much worried about maintenance, as her husband's position was altered by the War and he was trying to get out of regular payments; moreover, he was an "alien enemy."

Then thought he would want to "put her out of the way," and get rid of the maintenance order; that he caused a leak of gas in her bedroom; imagined she saw him when visiting a cathedral, and feared he would attack her, or that he might try to convict her of being a spy; became confused and excited; imagined signals for help were being made from a convent opposite; saw blue birds and a white bear on the carpet as an emblem of Russia. She recovered in a short time under the care of her sister.

Case XII.—Mrs. Q., aged 52. Sister depressed. Climacteric eighteen months ago. Husband was an artist and badly hit financially by the War, so that they did without servants, patient and her daughter doing the house-work. Then husband had appendicitis and patient nursed him through it until he was sent to hospital for operation. She became very fatigued, and her daughter becoming ill with pneumonia her energies were further exhausted; she became neurasthenic and sleepless, then lost interest, could do nothing, dreaded her house-work, and thought she was in jail. A definite attack of mental depression followed.

Case XIII.—C., aged 52, a man in business. No heredity, but subject to slight attacks of depression, especially when making up his annual balance sheet. At the end of 1915 he found he had made a loss of £34 on the year, and his men were called up for the War; became depressed, imagined he was ruined, and that he had ruined everyone and defrauded an insurance company; thought people said he was a thief, and wished a Zeppelin bomb would drop on him. He also accused himself of sexual irregularity in spite of being married, and there was probably some truth in this. He had to be certified.

Another circumstance to be taken into consideration is complete change of work or excess of work. Many educated women who have previously led quiet uneventful lives at home have suddenly felt it their duty to do "their bit"; have thrown themselves with energy into war work of various kinds—making splints and bandages, serving on committees, collecting money for various funds, joining ambulance or other corps, going as nurses, cooks, scullery maids or ward maids in temporary hospitals for the wounded, managing canteens for soldiers travelling from the Front (often until the small hours of the morning), and so on. Others doing regular work as clerks have suddenly had to increase the hours of work and to be "rushing all day."

To many healthy women, who have had very easy or idle lives before, work of such kind, if carried out with due regard to working within the limits of strength, has been of the greatest value; but to the neurotic or those who have previously had mental disorder there has been the danger of over-enthusiasm and a kind of emotional exaltation leading to work beyond the capacity, and overdrawing the fund of energy with resultant collapse.

In the case of men there has been sudden change of work from quiet civilian occupations to drilling, trench-digging, night work on anti-aircraft duty or as special constables, in addition to their ordinary work; and in the case of those joining the New Army a complete revolution in hours, work, companionship and prospects. It has not been my lot to come across cases of industrial fatigue in munition workers.

Especially in those aged over 40, who have thought it their duty to do something very active for the country, the effect of extra or unusual work has in my experience been detrimental, such men often becoming quickly fatigued, sleepless, irritable, depressed and suicidal, with delusions of wickedness, self-accusation of being a shirker or useless, or else suspicion of being thought to be so by those in authority. In some exhaustion of the neurasthenic or psychasthenic type, in others definite acute psychoses have developed.

Country gentlemen performing judicial functions have at times broken down from the added stress of extra work on tribunals, or of coming to decisions in the case of persons suspected of being spies, and have féared they have made wrong decisions, and that in consequence they were followed by spies or avengers.

Case XIV.—Miss R., aged 45; at the climacteric. Father had had a mental breakdown. Patient was a correspondence clerk, knowing French and German well. When the War began it "seemed to collapse her." At first it was thought the firm would have to close business, then suddenly they began to do a much larger business with France and Russia; she had to work much longer hours, and described it as "rushing all day." She began to sleep badly, waking after about three hours; had bad dreams; became depressed, restless and groaning when away from the office, and was unable to control it. As her means were small she feared losing her place, and so continued to work in this over-tired condition, lost a stone in weight, and became incapable of continuing with her work. She also was worried about a brother in the Army. Eventually she became certifiable, but her relatives did not take steps to put her under care.

Case XV.—S., a single man, aged 50. Had been in a quiet business in the country, which was not doing very well. No heredity, but he had had depression following influenza twenty years before. On the outbreak of War he joined an Ambulance Corps, then became recruiting officer to a drilling corps, worried himself about it, quickly became fatigued from constant drill; attention and volition failed, he became sleepless and depressed, and feared he might shoot others or commit suicide. He became quite unfit to do the work and was obliged to go under borderland care.

Case XVI.—Mrs. V., aged 42. Father died insane. She herself had had two previous attacks of puerperal insanity and two of insanity during pregnancy, and two others independent of the puerperal state, the last being in

1913. Early in 1915 she began to do voluntary work at a War Supply Depot; threw herself into it with enthusiasm; quickly became fatigued, then depressed, silent, vacant, resistive and negative. She quickly however, improved with complete rest in bed, and it is to be hoped refrained from joining other Committees.

Case XVII.—B., aged 32, a man of high intellectual attainments and unblemished moral character, engaged in teaching, enlisted as a private at the beginning of the War, but had not served abroad. No doubt he was associated in camp with very uncongenial and undesirable companions. He became at first physically, then mentally tired and unable to concentrate his attention, neglected tidiness in his uniform, was unable to keep his equipment in order, became introspective and depressed, and thought that drums he heard pointed to his funeral; said there was only one thing to do—that was to humiliate himself by committing sodomy, and thought of committing suicide. He had to be discharged from military duty and at once began to show improvement. Doubtless there was, in his case, a suppressed homosexuality.

Case XVIII.—P., aged 51. He himself was subject to depression. Was leading a quiet country life at the beginning of the War, but then rejoined his old Militia regiment, and his son went to France in November, 1914. He became worried: talked of nothing but the War; was sleepless, but was determined to go on working, although advised not to undertake physical exertion. Rapidly became worse, and, after resigning his commission under advice, declared it was a mistake and that people thought he was a fraud, became agitated and suicidal, and had to be certified. He recovered after some months under care.

Case XIX.—S., aged 45. Aunt insane. Always more or less an invalid: appendix removed ten years ago; had never done any military work before, but in August, 1914, joined the anti-aircraft force, was put on searchlight duty at night but was unable to sleep in the day, always felt tired and exhausted; then an attack of influenza made matters worse, he became profoundly neurasthenic, unable to give his attention or to do his business; refused to adopt any treatment involving rest; said he was in blank despair and talked of suicide.

Many other similar cases could be narrated.

Another group of cases is formed by those who, having foreign—in some instances German—names, have perhaps suffered from some real annoyance or persecution in consequence, or have recognized that their name and speech laid them open to the possibility of being looked upon as spies. Such persons have often suffered a good deal of misery, although perfectly friendly to this country, and it has been an easy step from the fear of being suspected to delusional belief that they are being watched or that the police are on their track—a definite psychosis developing with organized delusions of persecution.

Case XX.—A Swiss, aged 35, clerk in a foreign bank. Had had one attack of depression thirteen years before and was always liable to insomnia; had lived twenty years in this country. Having bought a Belgian Bond for a friend, inquiries were made by the Censor about the transaction, which was however approved. Patient began to think he had done some criminal act, imagined he had falsified the books at the bank, and that there was a conspiracy to make him out a criminal and a spy. He became sleepless, depressed, and suicidal, and had to be placed under care, and made no improvement.

Case XXI.—Mrs. W., aged 49, widow of a Scotchman, but was German-Swiss by birth. No heredity or previous attack, but was at the climacteric. She became depressed and sleepless, accused herself of being impure and unworthy, imagined people thought she was a spy because her sister had a German name and that she was to be arrested and would be executed; talked of suicide, had to be placed under care, then imagined that her sister was detained in the same hospital and was to be tortured and executed. She recovered after some months under care.

Case XXII.—Miss K., aged 28. Paternal aunt, suicide. Patient was an Alsatian and was living in England as a governess before the War. She had trouble with the police about her papers as she was Alsatian, also she could get no news of her parents, who were French, although living under German government. Her photograph had to be taken, and the photographer told her to fix her eyes on his during the process. She then imagined he had hypnotized her. She became depressed, sleepless, and suicidal, then confused and excited; thought she was conducting the War and telling General Joffre what to do; imagined she had been "taken advantage of" by a prominent Alsatian, and that intercourse had taken place from a far distance; imagined that although she was a Catholic she was to be sacrificed by marrying a Jew. She had to be placed under care and has remained insane, with hallucinations and delusions of persecution. She still thinks there is trouble about her papers and that some "noble Sir" is hostile to her. The case appears to be one of paranoid dementia præcox and the prognosis bad.

Although this country has been spared in great measure the horrors of war as experienced by the civilian populations of our Allies—the Belgians, French, Russians, Italians, Serbians and Montenegrins—in the invasion of their territories, yet it has had some experience of bombardment of coast towns and more especially of bombardment from the air by airships or aeroplanes in the different raids which have taken place at intervals.

It would appear to have been calculated by our enemies that the effect of raids by airships would produce a widespread terror in this country, and that the sight and rumours of burning towns, destroyed dockyards, ruined homes, and killed or injured civilians, would lead to clamours for submission and peace. It may be said, however, that

although in many instances the effects on individuals have been very serious, the total effect on the population has been very slight. In most of the raids the people, instead of rushing to earth and burrowing in cellars, have flocked into the streets or stood on the door-steps watching the unusual and interesting sight of one or more airships lighted up by search-lights and with shells bursting in the air around them, so that one has heard remarks as if the show were intended for the special benefit or amusement of the observers.

A friend wrote to me in July last: "The first raid was quite exciting, but many people rather enjoyed it. The damage was, except for the burning of a store and some unfortunate loss of life, quite unimportant, although the streets next morning had, owing to the large quantities of broken glass, a most dissipated appearance."

The main result of the raid of September 2, 1916, seems to have been to provide the people of London with intense satisfaction in the sight of the burning airship and the opportunity the next day for what a daily paper described as "beyond doubt the greatest free show that London has ever enjoyed." But of course where houses have been wrecked, or relatives killed or injured, there has not only been mental distress or anguish, but in a minor degree there has been something approaching the "shell shock" which has been such a feature in the cases of our soldiers suffering from the effect of intense and constant bombardment at the Front. Cases have been recorded of old people dying suddenly from the shock of the explosion.

Patients have spoken of being "stunned" by the noise, and when the explosion has taken place in or close to a house, the inmates have no doubt been profoundly shaken, if not otherwise hurt. A lady whose house was in the middle of the area affected by one of the raids in October, 1915, and all of whose windows were broken, told me she could not have believed that it was possible there should be such a noise and that the noise seemed to remain in her head for days. In that district houses were wrecked, holes were made in the ground, and people were killed, but her first action was not to hide in her cellar, but to go at once, although it was midnight, to the house of her son close by to see if he and his family were safe, and to save them the anxiety of wondering if she were all right. No doubt she is merely one example of the failure of "frightfulness" to have the effect desired by its authors.

In the October, 1915, raid on London, in common with many others, I was drawn by curiosity to watch the airship through field

glasses until it went out of sight, but two nights afterwards I dreamt most vividly that there were several large airships over the Langham Hotel, and that the latter was in flames. Another night I dreamt that there was a raid and that I was endeavouring to wake my household by hammering on their doors to induce them to take refuge on the ground floor but was quite unable to wake them.

In many cases apprehensiveness of further raids has led to insomnia or to vigilant sitting up at night to the early hours of the morning so as to be prepared, the effect being excessive fatigue or exhaustion. And even when the airship has not actually been seen the noise of distant firing, the blowing of hooters, or extinction of lights, have been enough in some cases to cause considerable fear with subsequent mental disorder. The likelihood of this result has been much increased by any previous attack of insanity, by pre-existing ill-health, or advancing age. The following are illustrative cases:—

Case XXIII.—A clergyman, aged 51, who had two previous attacks of maniacal excitement necessitating asylum care, was much alarmed by the air raid on London in October, 1915. Within two days he had become restless and excited, and sleepless, kept on saying "mad, mad," was grimacing, stripping off his clothes, impulsively violent, and hearing messages from God; and had to be put under care again, but quickly recovered.

Case XXIV.—Mrs. E., aged 36, always neurotic, and had had an attack of depression following lactation, leaving her unstable and subject to recurrent depression. She was staying at a coast town with relations for convalescence when an air raid took place there, bombs dropping close to the house she was in. She at once began to write to her husband and all her friends about insuring against raids, wanted to know if all the women were safe, became exalted and excited, and full of schemes, and had to be certified; she is still under care.

Case XXV.—Mrs. J., aged 60; daughter very neurotic, and she herself had had puerperal insanity thirty years before. While recovering from influenza and bronchitis she was much disturbed by the air raids of September and October, 1915. In the first one bombs fell near her house, and in the second she was alarmed by hearing guns fired. She became acutely depressed, feared to be left alone, was sleepless, constantly dwelt on the idea of disasters ahead, could not feed herself, and was suicidal, and had to be placed under care; she rapidly became worse and died.

Case XXVI.—Mrs. L., aged 35. Father alcoholic; no previous attack. In the summer of 1915 she had become over-tired as the result of moving house, and had had no summer holiday in 1914 or 1915 owing to the War and consequent upset of her husband's business. Then at the time of the October, 1915, raid, which took place in the near neighbourhood, although she did not

see an airship she felt "scared and frightened" by the noise of the hooters and firing, and saw shells bursting in the air. She complained of feeling dazed and "stunned," and as if in a dream or muddle, could not manage house or children; cried whenever her husband went out of the house, could not be left alone, and felt she must at once move from the neighbourhood. She had made considerable progress when I saw her and appeared to be on the way to recovery.

Case XXVII.—Mrs. G., aged 31. Seen at the Out-patient's Department, St. Thomas's Hospital. Mother alcoholic. Four years ago patient had been in Horton Asylum with depression following lactation, and was ill for eleven months. At the time of the air raid in September, 1915, her husband (a policeman) was on night duty; she saw and heard the airship, and for a month after that did not go to bed till 2 a.m. or later, often getting only three hours' sleep, and was apprehensive of raids. She became depressed again and quite unable to do her house-work, and voluntarily gave herself up to the Relieving Officer for Infirmary care.

Case XXVIII.—Mrs. M., aged 26; also seen at St. Thomas's Hospital. No heredity and no previous attack. Husband, a reservist, called up at the beginning of the War and serving in France. She had been very anxious about him. She was much disturbed by the October, 1915, raid, and by reading descriptions of other raids. She began to suffer from attacks of fear, associated with constant shaking, complained of trembling whenever she thought, had nausea and faintness, pains in the head and numbness after the fear, but no further mental disorder developed.

Case XXIX.—Mrs. S., aged 51, a widow. Husband dead eighteen years. Seen at St. Thomas's Hospital. Mother had been at Cane Hill Asylum, and patient herself had had an attack of depression seven years before. In September, 1915, a bomb dropped from an airship into the next street. She was very frightened and shaking at the time. Since then always apprehensive, especially on hearing any firing, sitting up to 4 a.m. when warned of a possible raid; sleep had been deficient and very irregular, generally about two hours; depression supervened, with desire to commit suicide. Her work involved her being alone all day. She improved when removed from the neighbourhood and staying with friends.

Case XXX.—Miss P., aged 38. Seen at St. Thomas's Hospital. No heredity and no previous attack. In the October, 1915, raid she saw the airship in the sky and heard the firing, sleep became very disturbed, and she was very apprehensive of further raids. At Easter, 1916, she again saw an airship in the sky, but no bomb fell near her. Her sleep became worse; she imagined that lights were thrown on her face directly she went to sleep, and that the "light seemed to wake her." She dreamt much about airships; then began to think that crowds followed her everywhere and intended to set fire to the house; heard them shouting and accusing her of immorality. She had to be sent to the infirmary and recovered.

Case XXXI.—P., aged 34. An early case of general paralysis, who had been found to have lymphocytosis of cerebrospinal fluid and positive Wassermann reaction. Had begun to fail in memory and in other ways before the declaration of War. Directly the War broke out, and before there had been any definite raids, he imagined he had important information to impart to the War Office, and wrote as follows: "I am writing just a line to say that it was quite evident from a listener's point of view that several Zeppelins were again over London yesterday. They came and went, concealed by the same old clouds, and diffused into the rain water and atmosphere the germs of syphilis; this in order to decay the strength of the nation. I have previously reported this to you and have had my evidence backed up by expert medical and scientific men of note. Every German carries '606' with him in his rations, and we ought to do the same. Why not send something up one evening when those clouds appear? I would give ample warning. You ought to act immediately, and I earnestly hope you will take steps at once to stop this dastardly plot."

In the old statistical tables of the Lunacy Commissioners there was in the list of causes a heading, "Fright and Nervous Shock," under which very small percentages were put down every year. That heading has disappeared in the present tables, being merged into "Sudden Mental Stress," otherwise it would have been interesting to see whether there has been a marked increase of cases attributed to fright due to the air raids.

Dr. William McDougall, in his work "Social Psychology," under the heading of "The Instinct of Flight and the Emotion of Fear," writes as follows: "Terror, the most intense degree of this emotion, may involve so great a disturbance both in men and animals as to defeat the ends of instinct by inducing general convulsions and even death;" and, "Fear, whether its impulse be to flight or to concealment, is characterized by the fact that its excitement more than that of any other instinct tends to bring to an end at once all other mental activity, riveting the attention upon its object to the exclusion of all others, owing probably to the extreme concentration of attention as well as to the violence of the emotion, the excitement of this instinct makes a deep and lasting impression on the mind." "Fear once roused haunts the mind; it comes back alike in dreams and in waking life, bringing with it vivid memories of the terrifying impression." He also points to the close relation of the "instinct of curiosity and the emotion of wonder" to the "instinct of flight and the emotion of fear," the difference between the excitants of curiosity and those of fear being mainly one of degree. No doubt those who have seen and watched airships from a safe distance have shown mainly the instinct of curiosity,

while those who have been unfortunate enough to be in the immediate neighbourhood of the dangerous effects of the bombardment have had more reason for the instinct of flight and concealment.

In some cases I have seen, the effect of a voyage in which there was fear of submarine attacks has appeared to precipitate a mental breakdown.

Case XXXII.—Mrs. M., aged 48. Father committed suicide. She had suffered from insanity in 1901, and was now at the climacteric. She went to Ireland on February 12, 1915, to bring her daughter home for her confinement. The "blockade of England" had been declared for February 18, and she became very apprehensive of the danger of the crossing from Ireland to England, lost her sleep, felt she must come back before February 18, and so had to cross in a hurry and before proper arrangements for her daughter were made. She rapidly passed into acute depression, said she was only a piece of wood and her arms and legs moving, that there was no peace and no hope for her, thought people would suffocate her and put an end to her, and was always expecting some terrible end.

Case XXXIII.—A., a farmer, aged 54. No definite heredity; always impulsive and bad-tempered. Very pronounced arteriosclerosis. His son was a "ne'er-do-well," who had run away from school twice and had stolen a motor cycle while in a training corps. Patient took him to an overseas dominion; the ship was followed by submarines and had to take a zig-zag course; it also took on board passengers whose ship had been torpedoed. While abroad patient had long journeys involving many nights in the train. On the voyage home they were again chased by submarines, and the passengers all lay down in their clothes with life-belts on. Sleep was much disturbed and there was constant apprehensiveness of danger. On returning home patient complained of loss of weight and loss of memory; was excitable, constantly talking, changing from one subject to another; became impulsive to his wife, but improved with rest and sedatives.

General paralysis, in several civilian cases I have seen, has appeared either to have been precipitated in its onset by the excitements or stresses of the War, or else the War has affected the mental aspect of the patient.

I have already mentioned the case where the patient's exaltation showed itself in imaginary knowledge about airships.

Case XXXIV.—J., aged 47, married, no children, no inheritance; some excess of alcohol; syphilis denied. He had been doing contractor's work for the Government and working very hard; then became exalted, magnified the extent of his contracts, thought he had made huge profits for his firm although he had really made stupid mistakes and lost money and his work had to be repudiated. Then fourteen days before I saw him he had had a fit with slight left hemiplegia, had affection of speech, absent knee-jerks; became blank and

forgetful but felt perfectly well, and talked of his large profits and the importance of his business.

Case XXXV. — L., aged 52, married; Jewish, of German birth but naturalized, and living many years in this country. There was a history of perforating ulcer of the foot, absent knee-jerks, and fixed pupils, for many years; and he had, at the age of 23, been under the treatment of Sir Jonathan Hutchinson, who was said to have given the opinion that he might marry subsequently. No doubt there had been syphilis. He lost his post in a bank owing to the War, became worried and sleepless, accused himself of sexual irregularities; went to the City and burnt trust documents belonging to others; became depressed and agitated, thought that he and his wife (also a German) would be taken to death, said he was a criminal and would be hanged; then became hilarious and talkative and forgot about his depression. Was found to have unequal and fixed pupils, absent knee-jerks, and speech affection. He was placed under care, quickly passed into an advanced stage, and died.

In two other cases of general paralysis where there had been gradual mental failure for many months, the onset of the War led to refusal of food, the patients insisting that as the War had broken out there was no food to be got in England. Both cases were of the depressed and hypochondriacal type of general paralysis.

In the absence, hitherto, of publication of the Annual Report of the Board of Control for England and Wales for the year 1915 it is not possible to give their figures, showing whether the effect of the War on the civilian population has been to cause an increase or diminution of insanity. The fact that some fourteen of the County and Borough Asylums in England and Wales have been turned into military hospitals for the accommodation of the wounded, and that arrangements have been made for the treatment of many of those soldiers who have broken down mentally while at the Front or at home, in hospitals under the control of the Army Medical Department and not in recognized asylums, at any rate while the cases seem curable, will no doubt have an important effect on statistics. There are also such questions to be considered as the restriction on the sale of alcohol, the large amount of money circulating in separation allowances, in money earned by women in munition factories and in other occupations formerly employing men almost exclusively, and the field of useful work which has been thrown open to many of those leading conventional or idle lives before the War, and who, therefore, have found their time occupied in "doing their bit" for the country.

It is far too early to gauge the full effect of these factors. I have, however, kindly been permitted to see the Report of the Asylums and

Mental Deficiency Committee of the London County Council for the year ending March 31, 1916, from which the following is an extract. The figures refer to the number of patients under care on January 1, 1916: "For the first time since the Council has been the responsible authority decreases are recorded both in the number of patients under reception orders—i.e., patients for whom the County is responsible to find accommodation—and in the total number of lunatics, the decreases being 637 and 950 respectively. The numbers chargeable to the Prison Commissioners (the lowest figure since 1890), on private list, and in workhouses, or with friends, have all decreased, while the number in the Metropolitan Asylums Board's institutions is less by 300 than on January 1, 1915."

I have also, through the kindness of Dr. John Macpherson, been able to see a copy of the Second Annual Report of the General Board of Control for Scotland, dated February, 1916. On January 1, 1916, there were in Scotland 19,108 insane persons of whom the Board had official cognizance, as compared with 19,557 a year previously, a decrease of 449. Deducting 317 children transferred from the Lunacy Acts to the Mental Deficiency Act, there still remains an actual decrease of 129 patients. The following sentence appears in the Report: "For the first time since the institution of the General Board of Commissioners in Lunacy in 1857, we have this year to record an absolute decrease in the number of all classes of the insane in Scotland." The total number of patients admitted to establishments during 1915 was 148 less than in the previous year, and 75 less than in 1913.

In the Annual Report of the Belfast District Lunatic Asylum for the year 1915, Dr. William Graham comments on the fact that the admissions, although showing a slight increase for the year compared with the previous year, "show a marked diminution as compared with the average for the past ten years, and the interesting and at first sight paradoxical fact is that this diminution takes place at a time when we are involved in the greatest war in the history of the world." He further says, "the fact is indisputable that insanity, like crime, has lessened during the period of the War," and that the greatest reduction is among women. He expresses the hope that "especially, though not exclusively, among women we shall witness a great diminution in those neurotic disorders that form part of the general problem of mental abnormality." "Among the mighty sociological factors which the present world-conflict has set in motion, not least will be the new value set upon all sorts of good work, and the new dignity which will crown the worker."

It has been said that the strongest nerves will win the War, and certainly the nervous systems of those engaged in the actual fighting at the Front have been submitted to more strain in this than in any previous war recorded in history. The concentrated intensity of shell fire, machine-gun and rifle fire, the unceasing tremendous noise and concussion of bursting shells, the poisonous effects of gas-attacks, tear-shells, and other diabolical inventions, the projection of burning liquids, the alarming effects of attacks from the air, and the earthquake violence of exploding mines, not to mention the terribly destructive wounds inflicted by modern artillery, must have tried the nervous systems of our troops to the utmost. We have no means of comparing the total results of these factors upon our men with similar results on the enemy, but if the demeanour of those returned from the Front, and their heroic conduct in attacks on strongly fortified positions is anything to judge by, it may at least be said that they have not shown themselves in any way inferior to the opposing side, while the demeanour of the civilian population under the stress of such attacks as have been made upon them tends to show that the nation and the sister nations composing the British Empire are not the craven degenerates which the mistaken psychology of our opponents expected them to be. Moreover, the figures I have quoted from the Reports of the Board of Control for Scotland and the Asylums Committee of the London County Council, are at any rate of the deepest interest, and worthy of special attention whatever may be their ultimate significance.

As to the effect of the War on patients already in asylums it would seem that it has been small. The more intelligent patients who are able to read the daily papers have made very ordinary comments on its progress; the dements have paid little or no attention to it; the delusional cases have sometimes used it in elaboration of their delusions. Dr. Isabel Emslie writes, in the *Edinburgh Medical Journal*, May, 1915, that: "Judging from admissions to the Edinburgh Royal Mental Hospital, the War has not made any actual increase in insanity in civilians. Among those who have had previous attacks and those who are weak-minded or highly strung, in many cases the War played a part in psychoses, although not actually causing them."

Professor Meyer, of Königsberg, records in the *Archiv für Psychiatrie*, 1915, lv, p. 353, an observation on fifty-three patients already suffering from psychoses before the outbreak of the War, and shows that they only reacted to it in a very small degree, and concludes that the morbid egoism prevents any essential influence of the War on existing psychoses.

Bethlem Hospital has, of course, been in the near neighbourhood of some of the air raids, and on several occasions patients have had to be moved from the upper to the lower floors with a view to preventing possible injury in the event of bombs falling on the building; which, however, has never taken place. On none of these occasions has there been the least panic among the patients. On one occasion a dance was in progress, and Dr. Porter Phillips informs me that it had to be suddenly terminated on account of an impending air raid. The patients were removed to the lower wards and an impromptu concert was organized by the medical and nursing staff with great success.

In summarizing these cases and many others I have been struck by the large preponderance of the influence of heredity or previous attacks. In 61·25 per cent. of my cases there has either been heredity (of insanity, suicide, nervous breakdowns, or alcoholism) or a previous attack; in 37·5 per cent. there has been heredity, and in 41·25 per cent. previous attacks, and in 17 per cent. heredity and previous attacks combined. In 48·75 per cent. the patient has been over 40 years of age, the average age of these being 50, and in 72·5 per cent. the patient has been over 30 years of age. Only six of the patients were 20 years of age or less. The sexes were about equally affected.

No doubt our colleagues who practise psycho-analysis will have noticed that there is a sexual factor in many of the cases I have referred to. In the cases of female patients there have been several with a history of divorce or separation from their husbands, either as the result of disagreement or unhappiness, or as the result of military or other service. There have been others who were widows and several at the climacteric. In others, again, there has been recent engagement or one-sided attachments. In the youngest case which I have seen, a girl aged 14, who had just arrived at puberty, there had been much sexual talk at a convent school she was at, about the invasion of Belgium and reports of rape and other horrors. She became excited, had the delusion that Germans were coming to attack her, was in great terror of everyone, mistook identities, thought doctors who went to see her were Germans, refused food, was noisy and screaming, and had to be put under care. A woman at the climacteric, long separated from her husband, placed her affection on our late Minister of War, saw his initial on her hand, felt they had community of feelings and ideas, thought she saw him in different disguises and that he intended to marry her. In the cases of men there have been such conditions as recent engagement in spite of previous mental disorder, sexual irregularities or unfaithfulness, and in some homosexual tendencies.

It would, of course, be wrong to ignore these conditions as associated factors in causing insanity, and in many of the cases there may have been repressed complexes. In such cases of mental disorder as appear to have been caused by the fear associated with air raids, however, I personally can see no object in hunting for a supposed phallic resemblance in airships. In many of the cases the airship has not been seen by the patient. In the *Review of Neurology and Psychiatry* for May, 1916, is an abstract of a paper by Buscaino and Coppola, published in the *Rivista di patologia nerv. e ment.*, xxi, and entitled "Disturbi mentali in tempo di guerra." The conclusion of the writers derived from experience in Professor Tanzi's clinique at Florence is that mental disturbances which occur in war, both in the Army and in the civil population, may be divided into two groups, in the first of which the mental syndrome is the direct and exclusive consequence of an external and physical factor, these being the less common. "In the second group, which is by far the most frequent, the syndrome is the result of the simultaneous action of two factors, or a complex of factors — one exogenous, physical or psychical, and the other endogenous—constituted by specific predisposition and not revealed by psychical manifestations."

It would appear that we arrive at the old and well-worn factors in insanity—heredity, previous attacks, and stress—and judging from my own limited experience and without the opportunity of considering statistics from the whole of the United Kingdom, it does not appear to me that the War is responsible for causing any great amount of new insanity. The stress or stresses have mainly affected those who were already prepared by heredity, previous attacks, or some predisposing instability, and who might have broken down in any case. On the other hand, in all probability in many cases tendencies which might have led to the development of neuroses or psycho-neuroses have been "sublimated" by useful work, and the final effect may be a strengthening of the mental constitution of the nation.

Section of Psychiatry.

President — Dr. R. Percy Smith.

(*January* 23, 1917.)

Morphological Investigations upon the Convolutional Pattern of Relative Brains in Man.[1]

By F. Sano, M.D.

Introduction.

"Wonder en is geen wonder."
—Simon Stevin.[2]

The problem of the inheritance of the convolutional pattern in man is far from being solved, and the time has certainly not yet arrived for testing the laws of Mendelian heredity on the basis material now to hand; nevertheless, large fields of statistical and biometric research still lie open for exploration in the near future. We must agree with the conclusion of Professor Sir W. Turner,[3] contained in his statement that "In each order the developmental process which determines the pattern of the cerebral cortex would seem to be regulated by the functional and physical necessities of the animals constituting the order, as well as by the conditions of hereditary descent." And we may expect the original pattern, proper to each order, to present a certain amount of variations, in accordance with racial, sexual and family peculiarities, although this

[1] From the Pathological Laboratory of the London County Asylums, Maudsley Hospital, Denmark Hill, London, S.E.

[2] Simon Stevin, born at Bruges, 1548, popularized the decimal system in the Seventeen Netherlands.

[3] W. Turner, "The Convolutions of the Brain," *Journ. of Anat. and Phys.*, 1890, xxv, p. 152.

has not been thoroughly proved to hold good for the convolutional pattern of the human brain. Moreover, "every furrow—even the sulcus centralis—" as Retzius [1] pointed out, "may be divided into two or more portions, and every furrow may also be connected with the neighbouring furrows." As an infolding of the brain wall at a certain point may be due to the pressure exerted on another point at some distance, "it is not necessarily the change in its direct surroundings which makes a sulcus change, nor necessarily a constancy in its direct contiguities which makes it constant," writes Ariëns Kappers.[2]

These, and other indications, emphasize the complexity of the problem. Numerous factors influencing the points of similarity and dissimilarity in relative brains, will be considered and discussed, so that an attempt will be made to discover the best method of comparison.

Major F. W. Mott, F.R.S., has kindly placed at my disposal for this investigation, material which he has been collecting for more than ten years. He had already submitted to me for investigation the brains of identical twins, which formed the subject of a monograph, published in the *Philosophical Transactions of the Royal Society*. Some ten years ago, Major Mott[3] commenced the study of heredity in relation to insanity, by establishing a card system in the London County Asylums, his objects being threefold—viz., (1) to ascertain if there existed an hereditary resemblance in the convolutional pattern; (2) to ascertain whether certain types of insanity are more prone to be inherited than others, and, if so, whether they are repeated in successive generations; (3) to ascertain if in a family with an insane heredity there is a tendency for the onset of mental disease to occur at an earlier age in the offspring than in the parent (anticipation).

These questions have been dealt with in numerous papers published in the *Archives* of the Laboratory. The number of cards now collected amounts to over 5,000 and 200 brains have been collected. Among them are seventeen pairs of brains of near relatives. Two of these pairs of brains have been carefully described by Dr. Edgar Schuster[4] in the *Archives of Neurology and Psychiatry*, vol. vi, and

[1] G. Retzius, "Das Menschenhirn," Stockholm, 1896, p. 95.

[2] C. U. Ariëns Kappers, "Cerebral Localization and the Significance of Sulci," *Congress of Medicine*, Lond., 1913, p. 371.

[3] F. W. Mott, "The Neuropathic Inheritance," *Proc. Roy. Soc. Med.*, Lond., 1913, vi (Sect. of Psych.), p. 23.

[4] E. Schuster, "Hereditary Resemblance in the Fissures of the Cerebral Hemispheres," *Arch. of Neur. and Psych.*, 1914, vi.

now the Director, Major Mott, has very kindly placed at my disposal fifteen pairs of brains of relatives. This valuable material, which has taken him so long to collect, forms the basis of the investigations which I have carried out in the Laboratory of the London County Asylums, Maudsley Hospital, Denmark Hill. I desire to offer my grateful acknowledgement to the Asylums Committee of the L.C.C. for permission to work there, and especially to the Medical Research Committee for the liberal grant accorded to me, by which I have been enabled to carry on this research in these times of struggle and distress.

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(I) THE MATERIAL USED IN THIS STUDY.

Details as to the material used in this study are to be found in Table A.

The average age of the parents at the beginning of their mental illness is 59·3, that of the children 27·3. The children all anticipate the age of onset in the parents—viz., 43, 8, 25, 21, 17, 44, 50, 31, 49 years. The average difference between the age of the parents and that of the children at onset of the mental disorder is 32 years.

The average weight of the brain of the male parents is 1,136·6 grm.; that of the female parents, 1,032·6 grm.; that of the male children, 1,313·9; that of the female children, 1,181. These small weights are a result of the method of hardening. Brains which have been for a long time in formalin solution, under slow evaporation and the repeated addition of new solutions, lose a fair amount of their weight. Special attention has been paid to this influence. Only one child has a smaller brain than that of the father, it is the daughter B of the second pair. The difference is not great and the brain of the daughter has been four years longer in the hardening solution than the brain of

TABLE A.—DETAILS AS TO THE ORIGINS OF THE SIXTEEN PAIRS OF RELATIVE BRAINS.

No.		Relationship	Difference of age	Age at onset	Age at death	Diagnosis	Infectious complication	Weight of the brain before and after hardening (grammes)		Cause of death
								Before	After	
I	A	Father ...	43	68	69	Senile dementia	—	—	1,220	Pneumonia
	B	Son ...	...	25	27	Epileptic dementia	Tuberculosis	—	1,285	Phthisis (pulmonary)
II	A	Father ...	43	46	85	Paranoia	—	—	1,160	Senile decay
	B	Daughter ...	...	38	38	Dementia paranoia	Dysentery	—	1,090	Exhaustion
III	A	Father ...	37	53	68	Senile dementia	—	—	1,030	Arteriosclerosis
	B	Daughter ...	...	28	34	Imbecility	Dysentery	—	1,100	Dysentery
IV	A	Mother ...	29	61	73	Senile dementia	Rheumatism	—	1,000	Endocarditis.
							Syphilis (?)			
	B	Daughter ...	...	40	41	General paralysis	Syphilis	—	1,125	Exhaustion, general paralysis
V	A	Mother ...	28	35	47	Chronic mania	Tuberculosis	—	1,205	Tuberculosis
	B	Daughter ...	...	18	18	Adolescent insanity	Dysentery	—	1,210	Acute colitis
VI	A	Mother ...	31	67	67	Senile dementia	Dysentery	—	906	Senile decay
	B	Daughter ...	...	23	38	Dementia præcox	—	—	1,140	Mitral disease
VII	A	Mother ...	32	66	77	Chronic mania	—	1,085	935	Senile decay
	B	Daughter ...	...	16	50	Adolescent insanity	Rheumatism	1,385	1,421	Broncho-pneumonia
VIII	A	Mother ...	40	63	75	Chronic mania	Syphilis (?)	—	1,070	Cancer
	B	Son ...	...	32	32	Melancholia	Tuberculosis	—	1,370	Phthisis (pulmonary)
IX	A	Mother ...	41	75	75	Senile mania	Syphilis	—	1,080	Senile decay
	B	Son ...	...	26	36	Recurrent mania	Syphilis	—	1,285	Pneumonia
X	A	Brother ...	8	26	49	Primary dementia	—	1,385	1,480	Pneumonia
	B	Brother ...	...	34	40	General paralysis	Syphilis	—	1,480	General paralysis
XI	A	Brother ...	3	29	32	General paralysis	Syphilis	—	1,170	General paralysis
	B	Brother ...	...	28	29	General paralysis	Syphilis (?)	—	1,280	General paralysis
							Rheumatism			
XII	A	Sister ...	6	71	88	Senile melancholia	Rheumatism	—	1,350	Senile decay
	B	Sister ...	...	67	83	Senile melancholia	—	—	1,125	Senile decay
XIII	A	Sister ...	5	52	61	Delusional insanity	Tuberculosis	—	1,055	Pulmonary and int. tuberculosis
							—	—	1,250	Pneumonia
	B	Sister ...	...	20	58	Delusional insanity	Tuberculosis	1,385	1,305	Pulmonary and int. tuberculosis
XIV	A	Brother ...	5	29	30	Melancholia	—	—	1,060	Dysentery
							Dysentery	1,185	1,105	Pneumonia
	B	Sister ...	...	25	25	Mania	—	1,515	1,545	Pneumonia
XV	A	Sister ...	3	49	57	Alcoholic dementia	—			
	B	Brother ...	...	40	60	Alcoholic epileptic dementia	—	—	—	Stillborn
XVI	A	Brother ...	0	—	—	—	—	—	—	Stillborn
	B	Brother ...	...	—	—	—	—			

the father, which suggests a loss of weight of greater importance so far as the daughter's brain is concerned.

Degeneration brings about variability; we may expect degenerated children to have larger or smaller brains than their parents, but the smaller idiotic brains, as a rule, do not reach the asylums, where the material has been obtained. It is also possible that the parents, having a brain weight below the mean weight of the general population, contribute children with a higher brain weight, progressing towards the mean weight of the general population. Although the figures of the brain weights are not very accurate, owing to the differences of hardening, it is interesting to observe that the average comparisons are in accordance with the results of more extensive statistics. But compared with fresh brains, obtained in the same asylum population, there is an average loss of 100 grm. for each group.

In five cases alcoholism of the father seems to have been the origin of the neuropathic heredity; in only two cases syphilis of the parents is signalized, but in eight cases (out of thirty-two) and three of which are "query" cases, syphilis has been considered as being present. In six out of fifteen cases, there was a marked neuropathic heredity in the family. In twenty cases, out of thirty, an infectious disease existed during the mental condition (the final pneumonia has not been accounted for); five times tuberculosis, five times dysentery, four times infectious rheumatism; twice rheumatic fever had existed with "query" syphilis.

The parents generally become insane in old age, and five times out of nine the illness is described as an evident senile condition. Paranoia and chronic mania have been the other conditions present. A similarity of character is often to be found in the parents and the children. A paranoic father has a daughter suffering from dementia paranoides. But the demential condition of the children is generally more accentuated than that of the parents. Except for the senile dementia, the parents preserve better memory. The mental condition in brothers and sisters is very similar. A brother under the influence of tubercular infection becomes melancholic and imagines himself persecuted; his sister nearly at the same age, after confinement, starts a maniacal condition, rapidly ending in a demential state. Both die after one year's illness. There may be a difference between the two diagnoses, but it is obvious that the general pathological condition is the same in its origin, in the constitutional nature of the disease and the poor physical resistance, failing to prevent the onset of a rapid fatal exhaustion.

Three cases of general paralysis were observed on different occasions with symptoms of dementia præcox, thus making the diagnosis difficult in the early stage (No. X, B; XI, A; XI, B). In these cases the neuropathic heredity was heavy. In one case the brother suffered from a typical condition of dementia præcox (X, A). As regards the other cases the mental condition was exactly the same in brothers, in sisters or in mixed siblings.

In the first group (I to IX) relating to parents and children, we shall have to trace the brain pattern of the parents in the bigger and more complex pattern of the children. In the second group (X to XVI) relating to siblings[1], brothers and sisters, we shall endeavour to find out whether the pattern originating from a like origin has undergone variations or noticeable modifications, and if so, how these have been forthcoming.

For the purpose of reliable comparison it will be necessary to find out the relative proportion of the influence of brain weight and of the influence of the sex, and whether there is a difficulty in eliminating these factors in the demonstration of familial peculiarities.

(II) Questions on the Convolutional Pattern.

The nomenclature of Retzius has been followed in this paper; attention has been paid to the remarks of Edgar Schuster. For the occipital pole I have adopted the anterior occipital of Wernicke, and I think there are sufficient reasons for calling attention to an inferior transverse occipital on the inferior surface.

In the case of each pair of brains eighty-three questions came under consideration. The questions and answers have been carefully revised three times over on separate occasions. The complete review of eighty-three questions dealing with thirty-two brains (sixty-four hemispheres) thus require 5,312 answers and is a very lengthy process. Experience obtained by the way yields fresh ideas and in respect of some questions it cannot be denied that a certain factor of personal impressions had its influence, although the comparison resulted in a formula, which always closely approximates to the objective consideration of the facts.

All the answers to the questions mentioned in the following list

[1] Siblings = offsprings of a single pair of parents.

(Table B) had been given with the description of each pair in a special table, the number of a question referring to the number of the subsequent tables. A summary had been added to each description in accordance with Table C.

The general description of the brains was written when the answers to the questions were collected, the table of the answers suggesting more rapidly to the mind the main points to be discussed. Descriptions of the brains have been twice revised. Then the thirty-two brains have been inspected at the same time and each question in the series has been checked; especial attention has been given to those questions which suggested some doubt or discussion, from a morphological point of view. Characteristic parts of the brains have been drawn in projection figures by a control method, and in natural size.

It was only when this preliminary work was done, that the answers were worked out by statistical methods, as will be shown later on. But from that moment not a single further alteration in the tables was allowed, so as to prevent any personal bias influencing the statistical conclusions.

No questions have been put discussing the insula, as it was not advisable to destroy the valuable material, which still can be used for other research work. Should questions be put as to the insula and as to the Heschl convolutions, I suggest the following as the most practical, in my opinion:—

Is there a sulcus brevis accessorius insulæ? Referring to the existence of four distinct gyri brevi (Retzius).

Is the post-central sulcus insulæ divided or not? (How many pieces?)

Is the sulcus temporalis transversus divided or not?

The insula has a very constant configuration and the results of its exploration would remain practically without great importance, as far as I have learned from those brains where it was accessible. But in several hemispheres the insula could not be examined and therefore, and also for the sake of preservation of the material, I have purposely omitted the details as to this subject. Further questions however, can always be put. For example, I did not pay attention to the concealed parts of the opercula; and the operculum frontale seems to be of importance.

TABLE B.—QUESTIONS CONCERNING FISSURES AND SULCI.

fl and *c*

1	Fissura lateralis, number of anterior rami.	
2	Fissura lateralis, number of posterior rami.	
3	Sulcus centralis + (Anastomoses with)	sulcus præcentralis superior.
4		sulcus præcentralis inferior.
5		sulcus postcentralis superior.
6		sulcus postcentralis inferior.
7		sulcus subcentralis anterior.
8		sulcus subcentralis posterior.
9		fissura lateralis separately.
10	Sulcus centralis cuts superomesial border.	

Frontal lobe

11	Sulcus præcentralis superior +	sulcus præcentralis inferior.
12		sulcus frontalis superior.
13	Sulcus præcentralis superior divided into two sections.	
14	Sulcus præcentralis intermedius present.	
15	Sulcus præcentralis inferior + (Anastomoses with)	sulcus frontalis superior.
16		sulcus frontalis inferior.
17		fissura lateralis.
18		sulcus subcentralis anterior.
19		sulcus diagonalis.
20	Ramus horizontalis separate.	
21	Ramus horizontalis + sulcus frontalis medius.	
22	Sulcus diagonalis well (+) or badly (−) developed.	
23	Sulcus frontalis superior, number of segments.	
24	Sulcus frontalis superior, + sulcus frontalis medius.	
25	Sulcus frontalis mesialis, well developed.	
26	Sulcus frontalis medius, well developed.	
27	Sulcus frontalis medius, number of sections.	
28	Sulcus frontalis inferior, continuous.	
29	Sulcus frontalis inferior + sulcus diagonalis.	
30	Sulcus frontalis inferior + sulcus radiatus.	
31	Sulcus frontalis inferior + sulcus fronto-marginalis.	
32	Sulcus frontalis marginalis, number of sections.	
33	Sulcus rostralis superior.	
34	Sulcus rostralis medius.	
35	Sulcus rostralis inferior.	
36	Sulcus rostralis transversus anterior joining sulcus cinguli and antero-mesial border.	
37	Sulcus orbitalis saggittalis + sulcus orbitalis transversus.	
38	Sulcus orbitalis transversus, number of pieces.	
39	Sulcus olfactorius, well (+) or badly developed.	
40	Sulcus olfactorius, mesial (+) or lateral (−) direction.	

To each question the answer concerning the left hemisphere of the elder brain (A) is put first—let it be "yes" (+) for example; then comes the answer for the left hemisphere of the younger brain (B)—let it again be "yes"; then for the right A; then for the right B. If all the answers are positive, the formula obtained on this manner will be + + + +. If the conditions in brain A are totally different from those stated in brain B, the formula will be

TABLE B—(*continued*).

Parietal lobe	41	Sulcus postcentralis superior +	Sulcus postcentralis inferior.
	42		Sulcus interparietalis.
	43		Sulcus parietalis superior.
	44	Sulcus postcentralis inferior +	Sulcus interparietalis.
	45		Sulcus subcentralis posterior.
	46		Fissura lateralis.
	47	Sulcus interparietalis proprius, continuous.	
	48	Sulcus interparietalis proprius + (Anastomoses with)	ramus ascendens sulcus temporalis superior.
	49		ramus ascendens sulcus temporalis medius.
	50		sulcus intermedius primus.
	51		sulcus intermedius secundus.
	52		sulcus occipitalis transversus.
	53		sulcus parietalis superior.
	54	Sulcus parietalis superior independent.	
	55	Sulcus parietalis superior, number of sections.	
	56	Sulcus parietalis superior + sulcus præcunei.	
Temporal lobe	57	Sulcus temporalis superior	anterior interruption present.
	58		middle interruption present.
	59		posterior interruption present.
	60	Sulcus temporalis transversus joining sulcus temporalis superior.	
	61	Sulcus temporalis medius, number of sections.	
	62	Sulcus temporalis inferior, number of sections.	
Occipital lobe	63	Sulcus lingualis independent.	
	64	Sulcus occipitalis anterior present.	
	65	Arcus intercuneatus superficial.	
	66	Lobulus parieto-occipitalis present.	
	67	Sulcus occipitalis transversus inferior present.	
	68	Sulcus verticalis continuous with fissura retrocalcarina.	
	69	Sulcus lunatus present.	
	70	Sulcus paramesialis at the lateral surface.	
	71	Sulcus occipitalis medius (lateralis) + a sulcus temporalis.	
	72	Sulcus occipitalis medius continuous.	
	73	Sulcus occipitalis inferior independent.	
Limbic lobe	74	Sulcus rhinicus externus joining the fissura lateralis.	
	75	Sulcus rhinicus internus present.	
	76	Sulcus collateralis	temporal interruption present.
	77		fusiform interruption present.
	78		lingual interruption present.
	79	Isthmus lobuli limbici concealed.	
	80	Sulcus subparietalis, number of segments.	
	81	Sulcus cinguli, number of segments.	
	82	Sulcus cinguli + sulcus subparietalis.	
	83	Sulcus intralimbicus present.	

+ − + − or − + − +. If the condition in the left hemisphere of A is different from the condition existing in the three other hemispheres, the formula will be − + + + or + − − −. One gets very soon accustomed to such formulæ, and the addition of similar formulæ out of one table of answers is very easy.

TABLE C.—COMPARISON TABLE FOR HEREDITARY RESEMBLANCES IN THE FURROWS OF THE CEREBRAL HEMISPHERES.

A = the brain of the first-born individual; B = the brain of the younger. Number of questions, 83. + means "Yes"; − means "No."

Similarities.

1 Number of cases where the four hemispheres show the same condition.
2 Number of cases where three hemispheres show the same condition.
3 Number of cases where the two left hemispheres show the same condition.
4 Number of cases where the two right hemispheres show the same condition.

Similarities of Differentiation between the Left and the Right Hemisphere.

5 Cases in which occurs the formula (+ + − −) or (− − + +); the two left similar and the two right similar, but the right condition being different from the left condition.

Peculiarities in one Hemisphere.

6 Number of peculiarities special to left A (− + + +) or (+ − − −).
7 Number of peculiarities special to left B (+ − + +) or (− + − −).
8 Number of peculiarities special to right A (+ + − +) or (− − + −).
9 Number of peculiarities special to right B (+ + + −) or (− − − +).

Confirmed Dissimilarities.

10 Number of cases where left A is similar to right A and left B to right B (+ − + −) or (− + − +).

Whether there is Inversion of Similarity.

11 Number of cases where left A is similar to right B and right A to left B (+ − − +) or (− + + −).

Indexes.

12 Apparent index of similarity, first method $(1\times4)+(2\times3)+(5\times4)$: (10×4) in percentage.
13 Apparent index of similarity, second method $(2+3)$: (10×2) in percentage.
14 Apparent index of dissimilarity.
15 Remnant percentage of variabilities or incomplete similarities (peculiarities and inverted similarities).
16 Absolute index of comparison, first method.
17 Absolute index of comparison, second method.
18 Results of comparison with other brains.

TABLE D.—COMPARISON OF RIGHT AND LEFT HEMISPHERES IN THE SAME BRAIN.

1 Number of similarities for the questions concerning the lateral fissure and the central sulcus (10 questions).
2 *id.* for the frontal lobes (30 questions).
3 *id.* for the parietal lobes (16 questions).
4 *id.* for the temporal lobes (6 questions).
5 *id.* for the occipital lobes (11 questions).
6 *id.* for the limbic lobes (10 questions).
7 Total (83 questions).
8 Percentage of similarity for the brain.

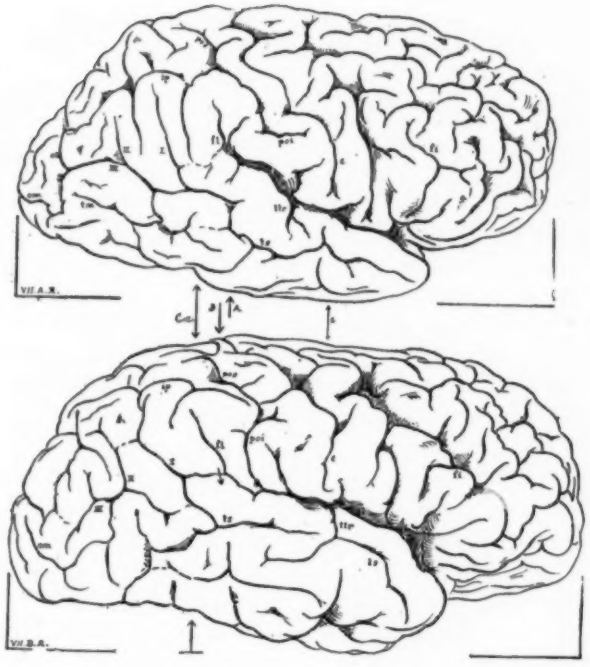


FIG. 1.

Lateral aspect of the two right hemispheres from brain pair VII (mother A and daughter B). In the mother's right hemisphere the inferior frontal sulcus (*fi*) is interrupted, in the daughter's brain it is not. In A the interparietal sulcus (*ip*) is not interrupted and joins the post-central superior (*pos*) and the post-central inferior (*poi*); in B *ip* is interrupted and does not join *pos* + *poi*. In A the superior temporal (*ts*) is not interrupted and does not anastomose with the transverse temporal (*ttr*), which is the case in B. It is the differences which strike the observer at first sight, similarities do not come into evidence so rapidly. A method therefore becomes necessary. These two brains VII A and VII B have but a small number of confirmed dissimilarities. C.c., end of the corpus callosum in both brains; B., superior end of the sulcus centralis in brain B; A, *id.* in brain A.; c, inferior end of sulcus centralis in both brains. The arrow in brain B near the lateral fissure (*fl*) indicates a deep annectent gyrus.

(III) First Results of Comparison. Indexes.

When the conclusions of the comparison of the twin brains were stated, I could not compare the results obtained with any others, as it was the first time that an attempt had been made to obtain a number expressing a degree of similarity for relative brains. Indications of any kind failed as to the value of each question for a comparative study, although the percentages of different authors as Retzius, Cunningham, and Eberstaller might have a real value for a general control. The papers of Waldeyer, Spitzka, Karplus and Schuster on relative brains do not arrive at a very definite stage of knowledge. By their most valuable work the problem is clearly put.

With a table summarizing the results, as I have given in my paper on the twin brains, it will be easier to check the actual value of the questions and to improve the method. Perhaps an attempt could be made to simplify the examination and to eliminate some questions, although this would only concern the brains now to hand. For a new series it always will be necessary first to propound a greater number of questions and to reduce their number later on, if it seems suitable or justified by the experience newly acquired.

The method I used for the twins consisted in adding the cases, where four hemispheres were similar, those where three hemispheres were similar and those where the hemispheres were similar to each other on both sides at the same time (Nos. 1 and 2 and 5 of Table C). The sum converted into percentages according to the number of possibilities of a complete resemblance is then divided by the percentage of complete dissimilarities. I have tried to simplify this procedure by taking the sum of the left similarities with the right similarities, reducing them to a percentage and dividing this percentage as in the former method, by the percentage of the complete dissimilarities. It is, of course, not necessary to reduce the numbers to percentages to obtain the index of comparison, but the percentages are necessary for the general consideration, being more easily understood.

The first method confers more importance on the conditions where three hemispheres are similar to each other, as it shows persistence of the common original type. Again attention must be called to our present ignorance as to the reciprocal value of each of the formulæ, which may occur. It seems justifiable to admit that a confirmed differentiation (+ + − − or − − + +) should be of more importance than a complete similarity in the four hemispheres (+ + + + or − − − −) because in

addition to a similarity there is a resemblance of differentiation between the left and right hemisphere, and moreover this is seen far more frequently in relative than in non-relative brains. But it would be premature to state any further estimate of its comparative value. Some indications on the other hand tend to suggest that it is better not to compare the four hemispheres in one formula as by the first method. The inheritance of the right pattern is somewhat different from the inheritance of the left pattern. The percentages of similarity for left and right hemispheres separately have been taken as a first element in the second method. The index of dissimilarity however comprehends the four hemispheres in one result, as it is the only manner of stating a real difference.

TABLE E.—COMPARATIVE INDEXES IN SERIES ACCORDING TO THE FIRST AND TO THE SECOND METHOD.

No. of the Series	No. of the brain pair	Absolute index, first method	No. of the brain pair	Absolute index, second method
1	XVI	5·65	XVI	4·87
2	XII	6·04	III	4·35
3	III	6·20	XII	5·54
4	IV	6·32	IV	5·60
5	IX	6·65	IX	6·20
6	XV	8·18	II	7·42
7	X	8·43	XV	7·50
8	II	9·21	X	7·93
9	VI	9·60	VI	9·00
10	XIV	11·14	XIV	9·70
11	VII	13·00	VII	11·40
12	XI	13·45	VIII	12·20
13	VIII	13·90	XI	12·30
14	V	13·90	V	12·40
15	I	13·95	I	12·60
16	XIII	14·00	XIII	13·00

N.B.—The differences are very slight and the index figures for the brain pairs remain nearly at the same places. Only in those for Case II is there a serious difference.

In order to facilitate the review of the cases I place them in series according to the two methods in Table E.

Female brains are slightly more similar to one another, about 1 per cent. more, than male brains. Mixed comparisons give the lowest numbers of comparative resemblance; the lessening of the numbers is about 11 per cent. It is to be noted that the brain weight of the females was 12 per cent. less than the average brain weight of the males. There seems to be a correlation between these differences; the disturbance brought about in the comparison by sexual difference seems to correspond to that percentage, but it is not the difference of brain weight only nor chiefly which determines the differences, as

TABLE F.—AVERAGES OF THE INDEXES OF COMPARISON ACCORDING TO SEXUAL INFLUENCE.

FEMALES COMPARED		MALES COMPARED		MIXED COMPARISONS	
First method	Second method	First method	Second method	First method	Second Method
6·02	5·54	5·65	4·87	6·20	5·35
6·32	5·60	8·43	7·93	8·18	7·50
9·60	9·00	13·45	12·30	6·65	6·20
13·00	11·40	13·95	12·60	9·21	7·42
13·90	12·40	—	—	11·14	9·70
14·00	13·00	—	—	13·90	12·20
Averages: 10·47	9·49	10·37	9·42	9·21	8·06

TABLE G.—AVERAGES IN DESCENDING PARENTAGE.

FATHER AND SON		FATHER AND DAUGHTER		MOTHER AND DAUGHTER		MOTHER AND SON	
First method	Second method	First method	Second method	First method	Second method	First method	Second method
13·95	12·60	9·21	7·42	6·32	5·60	13·90	12·20
—	—	6·20	5·35	13 90	12·40	6·65	6·20
—	—	—	—	9 60	9·00	—	—
—	—	—	—	13·00	11·40	—	—
Averages: 13·95	12·60	7·45	6·38	10·43	9·60	10·27	9·20

General average: First method, 10·30; second method, 9·13.

TABLE H.—AVERAGES IN COLLATERAL PARENTAGE.

BROTHER AND BROTHER		SISTER AND SISTER		SISTER AND BROTHER		MALE TWINS	
First method	Second method	First method	Second method	First method	Second method	First method	Second method
8·43	7·93	6·02	5·54	11·14	9·70	5·65	4·87
13·45	12·30	14·00	13·00	8 18	7·50	—	—
Averages: 10·66	9·91	10·01	9·27	10·60	8·35	5·65	4·87

General average: First method, 9·69; second method, 8·69.

will be proved in the chapter on sexual differences. Moreover the inherited family resemblance has a greater importance than the sexual differences.

The difference between descending and collateral parentage may be due to the fact that in the first group there are six (i.e., 33 per cent.) male and twelve (77 per cent.) female brains and four mixed comparisons (66 per cent.). In the second group there are eight (57 per cent.) male and six (43 per cent.) female brains and two mixed comparisons (28 per cent.). For a same degree of parentage very different numbers are forthcoming; no conclusions can be stated at present; the four cases of mother and daughter are especially interesting as they exhibit superior as well as inferior numbers.

The indexes obtained for each of the comparisons of relative brains have been put into Diagram I.

It is obvious that, the amount of similarities being nearly the same and varying only in one-seventh of the average number, whereas the number of complete dissimilarity varies in one-half of its own average, the resulting index of comparison is almost a mere function of the amount of dissimilarities.

As a control to those results I have made a great number of comparisons from some of the brains with *non-relative* brains. The next diagram (II) concerns the comparison between the father A of the first pair with his son and the thirty others not related to him, the comparison between the heaviest brain with thirty-one others and with the sister's brain, and the comparison between the negro's brain with thirty others and the mother's brain. The figures show that with respect to these comparisons neither the brain weight nor the sex exhibit an importance comparable to the personal and familial brain pattern. If the brain weight possessed an overwhelming importance, the series would come out according to the weight. If the sex possessed it the series would be according to the sex. If the racial influence had a real individual value, the negro and his mother would be at the beginning of the list, but they stand very far apart from each other. The relative brain of the son, however, comes out at the highest point of similarity with his father's brain.

More will be said about the influence of sex in a special chapter; as to the racial influence we can only review the question in brief, as only two negro brains are at our disposal. The conclusion arrived at in every anthropological research on the brain pattern has been that no peculiar condition whatever exists for one special human group. "Non conosciama

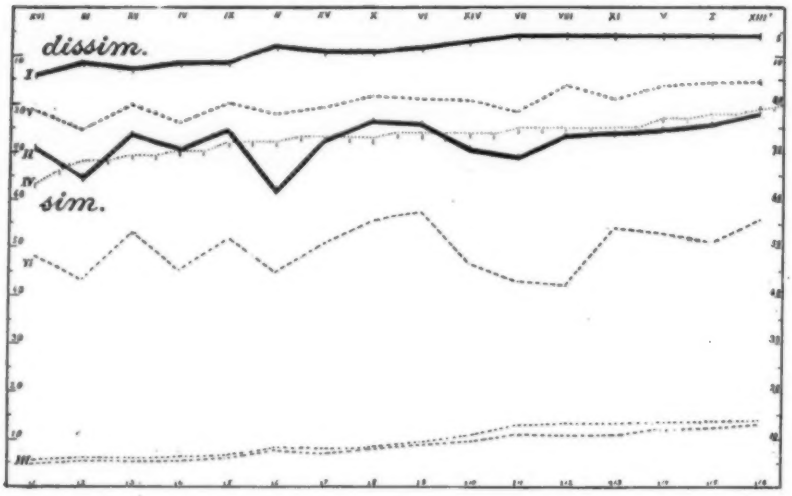


DIAGRAM I.

Comparison of relative brains. Seriation according to the absolute index of comparison.

(I) Indexes of apparent Dissimilarity (scale beginning from above).
(II) Indexes of apparent Similarity (second method).
(III) Absolute Indexes of Comparison (according to the two methods) (not in percentages).
(IV) Indexes of Similarity by comparison of hemispheres of the same brain.
(V) Indexes of apparent Similarity (first method).
(VI) Percentages of cases where the four hemispheres are similar (p. 44).

Scale beginning from below.

The space between the upper border of the diagram and the line (I) contains the complete dissimilarities; the space between (I) and (V) the peculiarities existing only in one out of four hemispheres and the cases of inverted similarity; the space between (V) and (II) contains the cases in which three hemispheres are similar; the space between (II) and the lower border of the diagram contains the similarities, left and right separately considered. Between (VI) and the lower border are the cases in which four hemispheres are similar. The Roman numbers on the upper border refer to the pairs of brains (p. 24). The numbers of the brains have not been given for comparison of hemispheres belonging to the same brain.

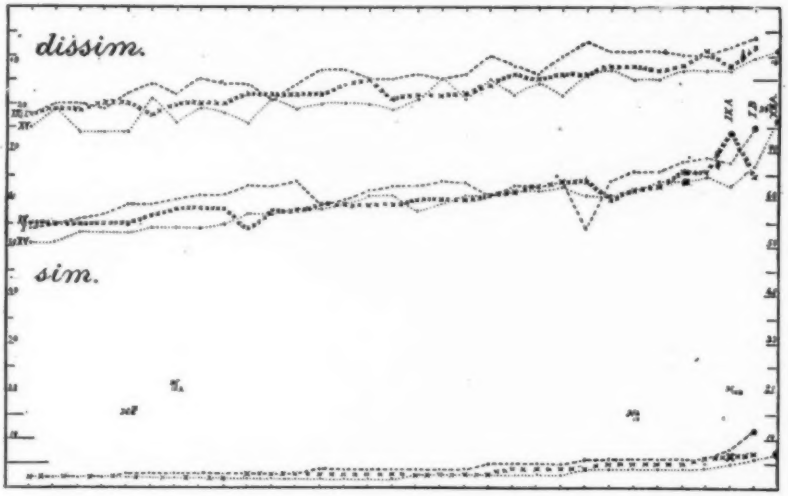


DIAGRAM II.

Comparison of non-relative brains, in series according to the absolute index of comparison :—

(I) Comparison of brain IA with thirty-one other brains, including the son's brain (IB).

(XV) Comparison of the heaviest brain (XVB) with thirty-one other brains and with the sister's brain (XVA).

(IX) Comparison of a negro brain (IXB) with thirty other brains and with the mother's brain (IXA).

The lines of dissimilarities with the scale to be read from above.
The lines of similarities (second method) with the scale from below.
The absolute indexes of comparison (no percentage).

The average percentages are :—

		IA	XVB	IXB
Non-relatives	Apparent similarity ...	62·29	58·91	59·95
	Incomplete similarity ...	2·40	3·13	3·24
	Peculiarities	20·66	18·53	19·30
	Complete dissimilarity ...	14·65	19·43	17·51

Non-relatives' total	Apparent similarity ...	60·380	Relative brains	73·0
	Incomplete similarity ...	2·923		1·9
	Peculiarities	19·500		16·9
	Complete dissimilarity ...	17·197		8·2

un attributo morfologico della solcatura cerebrale, che sia exclusivo di un dato gruppo umano," writes Sergio Sergi.[1] "Many variations indicate the constant tendency to oscillations and divergencies, but still more important seems to be the tendency to the preservation of some morphological characters in relation to the sexual difference, independent of any ethnical difference."

Those who arrived at too hasty a conclusion in an extremely positive manner were speedily contradicted, and Parker's statement: "The negro brain bears an unmistakably nearer relation to the ape type than does the white," did not meet with any confirmation; quite the contrary: and the valuable work of Kohlbrugge[2] on the Javanese brains is one proof more that every kind of peculiarity in the convolutional pattern is to be found in every human race.

At the first examination of the group of relative brains, I did not know that two of these brains belonged to negroes, and I did not find any condition which allowed for the suspicion. After having duly considered the problem on this occasion, I am led to conclude that, if racial differences exist, as we may expect, it is only the statistical method on a large scale that will be able to render us further help.

Four hundred and ninety-six comparisons are possible with the brains described. When, exceptionally, the index of comparison with a non-relative brain comes out higher than, or approximates to, that of the relative brains, the confirmed differentiations, always more numerous in the relative brains, indicate which are relatives and which not.

Index of Variability.—By adding the similarities and the dissimilarities we obtain the amount of fixed characteristics; the remainder is formed by incomplete similarities and by peculiarities; it gives the percentage of *variabilities*, or unfixed characteristics. In all these estimations we are only considering the inheritance of the pattern of *one* parent, which is a preliminary study to that of the combination of both parents' patterns; the material did not allow of the latter being made. The remainder now of so-called variable conditions is smaller in relatives than in non-relatives. The smallest remainder occurred for the first pair (George Smith and his son George Smith), then follows the Negro family, then two old sisters, two Hebrew brothers, the twins, and so on (Diagram III)).

[1] Sergio Sergi, "Contributo allo Studio del Lobo Frontale e Parietale nelle Razze umane," Ricerche, ecc., R. Universita di Roma, ecc., 1908, xiv, fasc. 1, 2.

[2] Kohlbrugge, "Die Gehirnfurchen der Javanen." Akademie der Wetenschappen, Amsterdam, 1906.

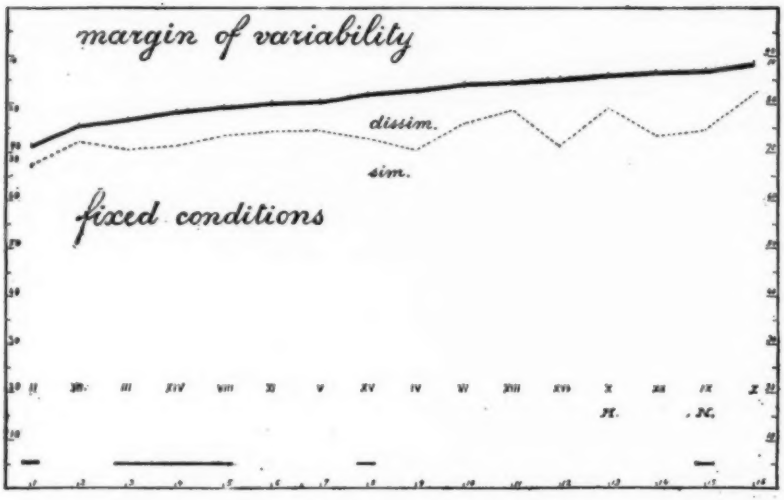


DIAGRAM III.

The margin of variability; seriation according to the index of variability (scale from above). The Roman numbers refer to the pairs of brains (p. 4). N IX are the Negroes, X the Hebrew brothers, XVI the twins. Comparisons of mixed sexes are underlined, according to the second method.

According to the first method the Negroes and the twins show the smallest figures for the remainder of variability. As we do not exactly know the racial origin of our subjects, it is not possible to control if the amount of variability increases with a more mixed racial origin.

There is a tendency for the sexual mixed comparisons to occur on one side of the diagram, and except for the Smith family (I) and the Negroes (IX), five brothers and sisters out of seven collateral comparisons are at the top of the percentage; the two others (brother and sister XIV and sister and brother XV) are mixed comparisons. Thus collateral parentage of the same sex shows less variabilities than descending parentage or than collateral parentage of different sexes.

Decreasing Similarity in Relative Hemispheres.—As proved in Diagram I, the apparent similarity between the two hemispheres of a brain is as great as the apparent similarity between the brains of two persons directly related by blood. The index between the two hemispheres of the first examined brain A, I (father) is 0·74. The index for

his left hemisphere compared with the left hemisphere of his son is also 0·74. The index of the father's *left* hemisphere with the *right* one of the son, however, is 0·70. The index of the father's left hemisphere compared with the left of a *non-relative* brain is 0·64, and with the right hemisphere of that non-relative brain is 0·59. As a non-relative brain I took the brain II, A. More examples are easily established by the aid of the tables annexed to each description of a pair of relative brains. This proves that there is a difference between left and right hemispheres *in man*, and that our left hemisphere more resembles the left than the right hemisphere of a non-relative brain. But as the resemblance of the left hemisphere to the right one of a relative brain is greater than that obtained with the right of a non-relative, it also proves that the familial type is to a certain degree common to the *two* hemispheres, and that there may be some Mendelian or Galtonic proportion between the three first terms of the decreasing similarity:

$$\underset{0\cdot74}{\frac{\text{Left father's h.}}{\text{Right father's h.}}} = \underset{0\cdot74}{\frac{\text{left father's h.}}{\text{left child's h.}}} > \underset{0\cdot70}{\frac{\text{left father's h.}}{\text{right child's h.}}} > \underset{0\cdot64}{\frac{\text{left father's h.}}{\text{left non.-rel. h.}}} > \underset{0\cdot59}{\frac{\text{left father's h.}}{\text{right non.-rel. h.}}}$$

The equality of the two first terms (0·74 and 0·74) cannot be considered as a case of homotyposis because the third term shows that the father's left hemisphere is not equally similar to the two child's hemispheres. Moreover the whole demonstration clearly shows that each hemisphere of the brain is a *differentiated* organ.

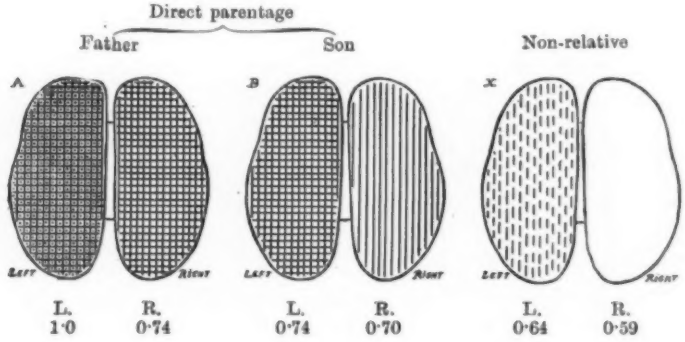
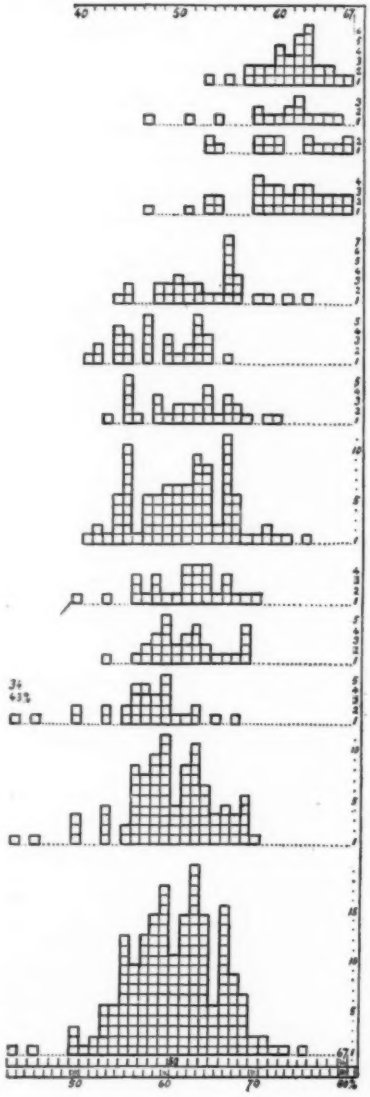


FIG. 2.

Decreasing similarity.



Comparison of

Hemispheres of the same brain.

Left relative hemispheres.

Right relative hemispheres.

Sum of the relative hemispheres.

Left I, A, with left non-relative hemispheres.

Left IX, B, with left non-relative hemispheres.

Left XV, B, with left non-relative hemispheres.

Sum of left non-relative hemispheres.

Right I, A, with right non-relative hemispheres.

Right IX, B, with right non-relative hemispheres.

Right XV, B, with right non-relative hemispheres.

Sum of right non-relative hemispheres.

Sum of left and right hemispheres.

DIAGRAM IV.

Frequency polygones. The diagram is composed of the original numbers of similarity, out of eighty-three questions. The corresponding scale of percentages is added at the lower border of the figure.

All the averages and indexes which I have given are arithmetical means, except the absolute Index of Comparison. They are not to be compared with the ratio or degree of correlation obtained by trigonometric calculations. For instance, when I found that the complete similarity for relative brains corresponds to 0·49, this is an arithmetical mean for the sixteen pairs of brains which I examined and cannot lay claim to be anything more. Some of my tables are capable of being put into frequency polygones, as in Diagram IV. The modal value and the frequency of the modal brains would appear to give a geometrical result, slightly different from the arithmetical result, but I do not think that we can yet calculate it with sufficient accuracy. Fresh indications which appear to be established by this method are as follows:—

(1) The negative standard deviation, indicating the progressive variations, is greater than the positive standard deviation, and this also appears from the figure giving the brain weights of males and females. It proves that the actual pattern of the human brain is forthcoming from a less constant and less similar pattern. It suggests that the ancestors of the present human race had a lower index of comparison than that to be found at present.

(2) The modal value of the similarity in relative brains seems to be a little less and behind the modal value of the similarity between hemispheres of the same brain.

(3) The curve of the totals for the left hemispheres has two summits, owing, as it will be proved further on, to the influence of sexual differences from unequal development of the speech centres, culminating in the left hemisphere.

Conclusions to the Third Chapter.

The comparison of relative brains has an individual value. In other words, when the convolutional pattern of a series of brains has been described, no indications have been forthcoming until now to determine which of them are the heaviest, nor whether a brain belongs to a male or to a female, nor to which family of the human race it is most likely to belong. But the pattern of a father's brain being given, it is possible to find his child's brain out of a series of non-relative brains.

One hemisphere of a human brain resembles the other hemisphere as much as it resembles the hemisphere of the same side in the parent, the child, or the sibling. But it less resembles the hemisphere of the other side in the family members of direct parentage; still less the

hemisphere of the same side in a non-relative brain, and least of all the hemisphere of the opposite side in a non-relative brain.

The constancy of an hereditary resemblance in the convolutional pattern is thus undoubtedly proved.

(IV) Discussion of the Questions.

The same summary (Table C) which has been used for each case of comparison can be used for each question. The figures obtained by this manner have been collected in tables, which show the amount of similarities and dissimilarities, and give an idea of the variability of each part of the convolutional pattern to which the question refers.

Table I.—Results summarized according to Table C, and Grouped for the Lobes of the Hemispheres in Percentages.

	Four similarities	Three similarities	Left similarities	Right similarities	Confirmed differentiation	Left peculiarities in A	Left peculiarities in B	Right peculiarities in A	Right peculiarities in B	Complete differences	Inverted similarity
	1	2	3	4	5	6	7	8	9	10	11
Fl and *c* ...	58	25	75	81	6	8	9	6	5	5	1·9
Frontal lobe ...	57	24	75	75	5	8	6	8	6	8	1·6
Parietal lobe	43	35	72	69	9	7	9	9	10	8	2·7
Temporal lobe	37	42	59	66	4	7	16	13	7	9	3·9
Occipital lobe	45	35	88	81	11	8	6	9	11	8	0·5
Limbic lobe ...	41	31	66	67	9	11	6	8	6	11	2·4
	49·9	30·8	72·8	73·1	7·2	8·5	8·2	9·0	7·9	8·1	1·9
			73·0			16·7		16·9			

Lobar Differences.—The central area and the frontal lobes are relatively very constant in their hereditary resemblance. The parietal lobe is a little less similar. The temporal lobe is the most variable, as a result of the differences in the auditory area, always more complex on the left side and showing leading conditions in that side, as it will be demonstrated further on. Peculiarities in the right temporal lobes are numerous, showing that when a condition exists in both hemispheres of one relative brain, this condition will exist on the left side of

the other brain, as a rule, rather than on the right side. The top of the temporal lobe is one of the latest developed and the most advanced extensions of the brain and therefore also the subject of greater variability. The conditions of the occipital lobes are less similar from left to right in the same brain than is the case for the frontal lobes (*see* Table I), but in each pair of left or right hemispheres they show the most constant similarity. The high figures in columns 3, 4 and 5 prove this. The questions relating to the limbic lobe only refer to its boundaries; as a matter of fact the limbic lobe is nearly identical in all the brains, but its boundaries are uninterrupted or deeply grooved in consequence of the pressure exerted by the development of the others, especially of the temporal lobe. The lower figures for the limbic lobe therefore do not possess that importance which they would have, were they to be found for the questions concerning the structure or form of the lobes themselves.

Some relative brains may be more like one another in the frontal, others in the occipital hemispheres. This also means that the variability may be more or less accentuated in the different lobes. It would be of great interest to follow up the special tendencies in more generations and to bring them into accordance with personal qualities or deficiencies.

(1) *Cases of Four Similarities.*—Questions giving a low number of four similarities relate to conditions undergoing great variability; questions giving a very high number are those referring to exceptional conditions, the rarity of which leave, for most of the brain pairs, a very great number of similarities, without particular significance. It would be of great value to discover conditions which should be exceptional, and at the same time constant in their hereditary transmission, but as far as my series is concerned there is only one example of this sort—namely, the interruption of the calcarine fissure (fig. 3); but there is no proof that this would be constant in other cases. There are 663 cases out of 1,328 giving four similarities—i.e., 49·9 per cent.

(2) *Cases of Three Similarities.*—The number of cases in which three hemispheres are similar is smaller than that in which the four hemispheres are similar, because three similarities mean one peculiarity, and peculiarities are rather exceptions, which exhibit variability. As a general rule it seems evident that the constancy in the brains examined corresponds roughly to 50 per cent. of the morphological characteristics and that the variability corresponds to 30 per cent. But it is also necessary to examine each hemisphere separately, with its homologue in the relative brain. There are 410 cases out of 1,328 giving three similarities—i.e., 30·8 per cent.

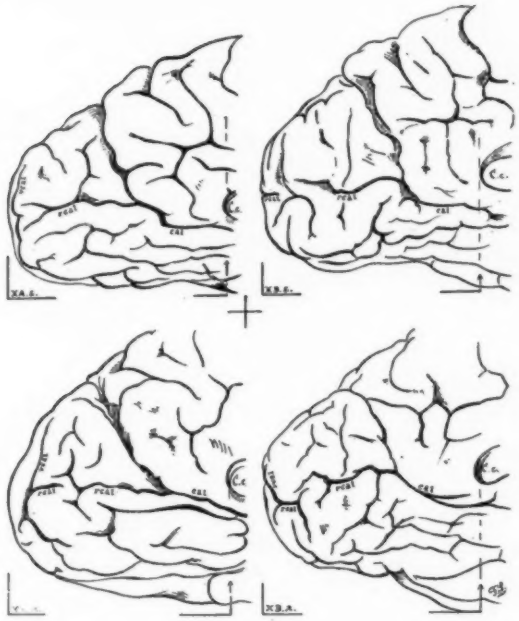


FIG. 3.

Interruption of the calcarine fissure. In the four hemispheres of the brain pair (X) the retrocalcarine fissure (*rcal*) was interrupted. The figures of the right occipital poles (mesial aspect) have been given in mirror, so as to make the comparison easy. *Cc*, corpus callosum; *cal*, stem of the calcarine fissure; *rcal*, retrocalcarine part; *vcal*, vertical part. There was no question in Table B about this exceptional condition. It gives one complete similarity more for the two Hebrew brothers.

(3 and 4) *Similarities on the Left and on the Right Hemispheres.*—The number of similarities on the left and on the right hemispheres is nearly the same. It is remarkable how closely the figures 968 and 971 approximate, notwithstanding the very different parts of the brain in which the similarities occur. It shows that there is a constant factor in the possibility of variations as well in the left as in the right hemisphere. The similarity for the whole brain gives 1,939 cases out of 2,656 possibilities—i.e., 73 per cent.

(5) *Confirmed Differentiation.*—The average of similarity being 73 per cent., and that of the confirmed differentiation 7 per cent., we may say that in one-tenth of the similarities there is a differentiation between left and right brain, maintained in the family. It would be

possible to a certain extent to recognize the brains of relatives by these conditions. Brain IX, B, has 12 per cent. such highly expressed similarities with the mother's brain, IX, A, and only two with brain IV, A, the most similar to it. Confirmed differentiations occur for fifty-one different questions, and in every part of the hemispheres. When they occur more than once for a question, the positive answers can be found as well on the right as on the left side; it means that no confirmed differentiation has a constant formula for a special question; for instance, a sulcus intracingulatus is to be found on both left hemispheres four times, with its absence on both right hemispheres, and twice on both right hemispheres, with its absence on the left hemispheres.

Exception is to be made for the question No. 60 (*ttr* + *ts*). This connexion never exists solely on the right side. When it is on the right it is also on the left. And when there is a confirmed differentiation, the connexion exists on both left hemispheres. That seems to be the result of differentiation in the left verbal auditory area.

(6 and 7) *Peculiarities on the Left Side.*—The results are:—

First group (descending heredity)	...	141 out of 797	=	17·6 per cent.
Second group (collateral heredity)	...	82 ,, 531	=	14·0 ,,
Totals	...	223 ,, 1,328	=	16·9 ,,

(8 and 9) *Peculiarities on the Right Side.*—The results are:—

First group (descending heredity)	...	136 out of 797	=	17·0 per cent.
Second group (collateral heredity)	...	89 ,, 531	=	14·0 ,,
Totals	...	225 ,, 1,328	=	16·9 ,,

Peculiarities mean as a rule variations on a pattern fixed in the three other hemispheres. The number of peculiarities occurring on the left side is nearly the same as it is on the right side. But taking the groups separately, in the left brain the children (B) show a very great variability, which the parents (A) show, but at a lower rate, in the right hemispheres. Seventy-seven questions give peculiarities on the left and seventy-six on the right side. Practically speaking, peculiarities may occur in every part of the convolutional pattern, but never exceed 17 per cent. of the possibilities. They are more frequent when children are compared with one of their parents, than when siblings are compared with one another. Left and right peculiarities together amount to 16 + 16 = 32 per cent., which is slightly higher than 30·8 found for No. 2 (three similarities); this

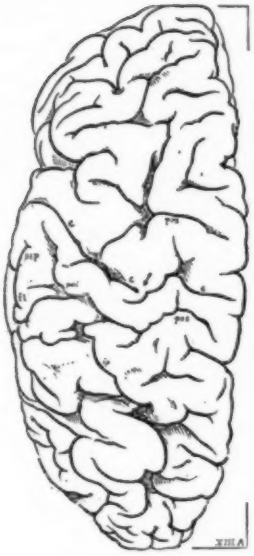


FIG. 4.

Interruption of the central sulcus. Left XIII, A, superior aspect, *c*, central sulcus (the arrow indicates its interruption); *prs*, sulcus præcentralis superior; *pos*, sulcus postcentralis superior; *poi*, sulcus postcentralis inferior; *ip*, sulcus interparietalis; *scp*, sulcus subcentralis posterior; *fl*, fissura lateralis. There was no question in Table B about this rare peculiarity.

results from some conditions, in which numbers have been taken instead of the signs plus and minus; more than one peculiarity have been obtained for the same question, where a case of three similarities was lost.

(10) *Complete Dissimilarity.*—One hundred and nine cases out of 1,328 give 8·2 per cent. of complete dissimilarities. The number of complete dissimilarities to be found in comparisons of six couples of different sexes (father and daughter, mother and son, sister and brother, i.e., 37·5 per cent. of the cases) corresponds to 40·1 per cent. of the total cases of complete dissimilarities. In ten couples of similar sexes, i.e., 62·5 per cent., the amount of dissimilarities is 59·8 per cent. The amount of dissimilarities is thus slightly greater for the group of dissimilar sexes. But sexual influence, as a rule, is not very marked. An exception seems to exist in the case of No. 24, where the difference is clearly in favour of the sexual influence (*see* § VII). For the questions 16 and 21 the influence of the brain weight is the only efficient factor. For question 35 brain weight and sex combine. The differences in cases of similar sex are directly determined by the weight of the brain and exist especially in the association areas. The development of the central region in a bigger brain brings the ramus horizontalis nearer to the sulcus frontalis medius, and by this means

effects their junction; moreover, the sulcus frontalis medius is better developed in bigger brains. The same result occurs in the case of the sulcus postcentralis superior, which joins the parietalis superior, and the latter, increasing in importance, is divided into two portions. In the lower part of the central region the same development again brings the sulcus diagonalis into closer connexion with the sulcus frontalis inferior, or the præcentralis inferior in conjunction with the frontalis inferior. A larger development of the frontal lobe brings about a good sulcus frontalis mesialis, or a good frontalis medius, sometimes both together. A third sulcus rostralis becomes more frequent (especially in males). The development of the temporal pole causes the interruptions of the superior temporal sulcus to be more frequent.

			Present	Index of similarity	Complete differences	Index of the question
59	Sulcus temporalis superior	Posterior interruption	7	0·84	—	27
58		Middle ,,	34	0·59	0·12	5
57		Anterior ,,	34	0·56	0·18	3

In the fœtal brain, the superior temporal sulcus develops first in its posterior part; its posterior interruption is the least frequent, and gives the least number of variabilities; then comes the middle interruption, and late in fœtal life, or even after birth, the anterior part develops, and therefore as variability has more chance of arising, *similarity decreases.*

Differences in the occipital lobe seem to be independent of sex, or brain weight, but are more under the influence of the family and individual complexity of the brain pattern.

(11) *Inverted Similarity.*—Inverted similarities, when the left condition of A resembles the right of B, and at the same time the right A resembles the left B, are rather exceptions in comparisons of relative brains. This rarity lends support to the conclusion that similarities and dissimilarities are facts of morphologic value. If they were results of pure hazard, without any relation to biological factors, inverted similarities would occur as much as confirmed dissimilarities and as total similarities. Before arriving at any conclusion as to any further significance of inverted similarities, it would be necessary to have the brains of both parents of the child examined. Only twenty-six cases out of 1,328 give inverted similarity, i.e., 1·9 per cent.

Reduction of the Questions.—An attempt may be made to reduce the number of the questions, to eliminate those which are of less value for the general description of the convolutional pattern. A number of these questions, referring to exceptional conditions, exercise only a moderate

influence upon the final result, and it may be assumed that they rather confuse it in an amount of indifferent answers. Therefore, I made some experiments, eliminating the questions which give fifteen and sixteen similarities in the hemispheres of the left or on the right side, eliminating also questions giving less than ten similarities on one side. The number of questions was thus reduced to forty-eight, but the indexes obtained were not satisfactory, and it was moreover evident that this method of performance did not eliminate a corresponding number of complete dissimilarities. It clearly appeared that for comparisons of non-relatives, the dissimilarities do not occur more frequently in the questions, which give a low number of similarities than in those giving a high number of similarities. I thus came to the conclusion that in the present conditions of our knowledge as to relative brains, *it is still better to increase than to reduce the number of the questions.*

(V) On Sexual Differences.

Up to now we have found two conditions in which sexual differences could be accounted for. There is the well-known influence on the brain weight. The average numbers were 1,297 grm. for the males and 1,130 grm. for the females. These figures are reduced by the method of hardening, but preserve a comparative value. For fifty-six male brains and fifty-six female brains, taken fresh as they were removed at the post-mortem room shortly after death, and coming from the same asylum population, I found the following numbers, carefully controlled: For the male brains, 1,385 grm.; for the female brains, 1,200 grm. For the series of the relative brains the figures do not extend much more for the males than for the females, but the numbers are low and different (twelve males, eighteen females). When larger numbers are taken, it appears that the variability of the weight is greater in males than it is in females.

A second difference was found for the connexion between the superior frontal sulcus and the middle frontal sulcus. In each of three cases the male had no anastomosis, where the female gave one on both frontal sides; a case of discrepancy occurred for the twin brains, where the bigger brain acts as a male and the smaller as a female, if one may say so. For thirty-two positive answers about this anastomosis (*fs* + *fm*), thirteen—i.e., 40·6 per cent.—belong to seventy-four—i.e., 43 per cent. males and nineteen—i.e., 59·3 per cent.—belong to eighteen —i.e., 56·2 per cent.—females, showing that indifferent types lessen the

special difference of well-accentuated types; but only one woman, XIII, B, has her two lobes free from this anastomosis, where five males (VIII, B; III, A; XIV A; XV, B; XVI, B), remain completely free from it. Although it might be considered as a result of a bigger brain, that the connexion is wanting in males, the case of III, B, the daughter, who had a bigger brain than her father, does not confirm that hypothesis, the father exhibiting the lack of anastomosis and the daughter not.

Measurements may show that the male brains have a comparatively broader frontal development, and thus explain the origin of this tendency to sexual difference. I have no figures for this control.

In four out of five cases the female brain showed lack of anastomosis in the right hemisphere. In four out of five cases the male brain showed lack of anastomosis in the left hemisphere. Thus the left hemisphere affords the best indications of this sexual difference. The male character exists in 64 per cent. of the male cases, the female character in 88 per cent. of the female cases. This difference is not confirmed by the percentages of Retzius, but the main sexual differences in Retzius's numbers also exist in the middle frontal sulcus, which is twice as much interrupted in males as in females. Possibly a difference of interpretation is the cause of our discrepancy, and thus the conclusions approximate to one another more than appears at first sight.

To discover further sexual differences it is advisable to check in the case of each question the number of positive answers in males as compared with those of the females. Cunningham thinks that the sulcus centralis begins with two portions, but that the sulcus præcentralis superior and the sulcus præcentralis inferior have a common origin and are separated by an annectent gyrus of later development. The figures of my series, although very small, lend support to that hypothesis, as the male brains, being larger and generally more evolved, show only 7 per cent. remnants of continuity, the female brains showing it in 22 per cent. of the cases. The figures of Retzius are very different, males showing 27 anastomoses per cent. and females only 21 per cent.; the right female hemispheres did not show any anastomosis. Nevertheless the average results of Retzius are still lower than those of Cunningham (32 per cent.), and Eberstaller (24 per cent.). Mine are the lowest, although the series relates to small brains and degenerated individuals, in the case of half of them at least. It is possible that racial differences intervene here. The ramus horizontalis of the frontalis medius remains more frequently isolated in males.

The two former conditions, separation of *prs* and *pri* and independence

of *h*, may be considered as the result of an increase of brain substance on the surface and as a consequence of the tendency of isolation of the sulci concerned. But the ultimate result of such an increase of cortex in the central region ends in the concealment of cortical substance, and the anastomosis of the sulci in other places, as it is generally admitted. One may also suggest that the concealment and the anastomosis indicate earlier maturation and fixation of primitive regions, which become fixed before the further development of the cortex allow the convexity of the gyri to extend more widely, and the sulci existing on their surface, such as *h*, to remain separate. Four connexions of the central sulcus and two more in the immediate boundaries of the central region point to a greater complexity of that part of the male brain from the increase of concealed cortex (4, 5, 8, 6). No. 15 is quite an exceptional condition, occurring in only one out of the sixty-nine hemispheres.

The sulci rostrales are more developed in the male brains and the fusiform hypocampic annectent gyrus also exists more frequently in these brains. It may be correlated with the physiology of smell and genetic associations.[1] In three female cases, and in the smallest of the twins, there was only one sulcus rostralis sagittalis.

TABLE J.—ANASTOMOSIS OF THE TRANSVERSE TEMPORAL WITH THE SUPERIOR TEMPORAL.

		L.	L.	R.	R.	
I	Father and son ...	+	+	−	−	both on left side.
II	Father and daughter	+	−	+	−	the father on both sides.
III	Father and daughter	−	+	−	−	the daughter on left side.
VII	Mother and daughter	−	+	−	+	the daughter on both sides.
VIII	Mother and son ...	+	+	−	+	son on both sides, mother on left side.
X	Brother and brother	+	+	−	+	B on both sides, A on left side.
XIV	Brother and sister ...	+	−	−	−	brother on left side.
XVI	Twin brothers ...	+	+	−	−	both on left side.
	Totals ...	6	6	1	3	

Males: Left, 9; right, 3. Females: Left 3; right 1.

The further extension of the temporal transverse sulcus and its connexion with the superior temporal gives the most remarkable figures, showing that this connexion exists more frequently in males, and in each sex three times more in the left hemisphere in relation, as we may understand, with the development of the præ-Sylvian region.

[1] This hypothesis was stated in writing, when Professor Springthorpe, of Melbourne, visited the laboratory and told me he had localized the cerebral genetic centre in the lower parts of the frontal lobes. (Handbook for Therapeutics, &c.)

Some closer details will therefore illustrate this question, the importance of which results from the fact that the transverse temporal is often only indicated in primates, and may be undoubtedly considered as a sign of higher evolution (*see* Table J).

This is a morphological demonstration of the localization of more evolved development in the specialized auditory region of the left hemisphere correlated with the physiology of speech, and it illustrates the general view of Turner, quoted in the introduction of this study.

Another proof is to be found in the comparison of the hemispheres of the same brain one with another. The comparisons have been summarized for each lobe in each brain. Male brains have been united in totals separately from the female brains. The result shows that there is a rate of differentiation between the left and right hemispheres in the same brain, nearly the same as the total variability between one family member and another. When, however, the differentiation is concentrated upon the conditions of one lobe, the other lobes remain more similar. In the male brain the differentiation exists, especially in the temporal and limbic lobes, but for the latter it is an indirect result of the development of the temporal cortex as the questions refer to the boundaries of the limbic lobe, which are under the direct influence of the temporal cortical development and pressure. The receptive area of speech associations being more developed in the left hemisphere in man, appears to be one of the main origins of this sexual difference.

An attempt has been made to discover whether some conditions show more constant similarity in females than in males, and vice versa, in other words, whether the mother is not prepotent for the female characteristics of her pattern, which is to be inherited by her daughter, &c. The figures were too small to allow any conclusion being drawn.

As far as I can gather, the typical male brain should have:—

(1) Well-developed speech centres: a broad frontal lobe.

(2) Three, at least two, sulci rostrales, on both sides; no connexion between *fs* and *fm* on the left hemisphere, at least; a well-formed *fms* or a good *fm*, sometimes both together on both sides; an interrupted *fi* in the left hemisphere; an anastomosis of *ttr* with *ts*, in the left hemisphere; and *rhi* joining *fl* and *col* in the left hemisphere.

(3) Comparing the hemispheres of the same brain with one another, the greater number of differences will occur in the speech centres in males, especially in the temporal lobe and its boundaries near the limbic lobe.

The typical female brain should show the contrary; with only one sulcus rostralis and connexion between the superior frontal (*fs*) and the middle frontal (*fm*), &c.

These indications do not permit of distinguishing with certainty a male from a female human brain, and the formula of their approximative value cannot yet be given.

(VI) The Results compared with those obtained by other Authors, and more Deductions.

If the object of these investigations were merely to demonstrate that there is a resemblance in the convolutional patterns in relative brains, they would even thus possess an interest as a formal proof of the morphological value of some details and peculiarities of the sulci and gyri. The first paper published by Karplus seems to have had that demonstration as its chief aim.[1] I could not obtain access to it in the libraries in London, and had to rely on Schuster's paper, which also proves the existing resemblance. Moreover, I possessed an abundant material, more interesting by itself than any bibliographical research, and only after having studied it thoroughly for some months I was inclined to review the bibliographical aspect of the question. My own work was then finished, and there was no possibility of further modification of it.

Spitzka was the first to describe three adult relative brains of brothers with precision and interesting details. He demonstrated their great resemblance and made some measurements, the most striking result of which shows that the corpus callosum had the same width, notwithstanding the differences in weight of the brains. In some of the brain pairs, which I have examined and which were available for such research, the same similarity exists.[2]

I am indebted to the kindness of Professor Ariëns Kappers, the Director of the Brain Institute at Amsterdam, for the opportunity of inspecting two papers, the contents of which I shall now discuss. In his second study on the inheritance and the variability of the convolutional pattern, Karplus calls attention to his former work.[3] He examined twenty pairs of relatives, five groups of three relatives, and

[1] Karplus, J. P. "Ueber Familienähnlichkeiten," &c., *Arb. Neur. Inst. Oberst.*, Wien, 1905, p. 1.

[2] Spitzka. "Hereditary Resemblance in the Brains of Three Brothers." *Amer. Anthrop.*, 1904, vi (to be found in the Library of the Royal Anthropological Society).

[3] Karplus, J. P. "Variabilität u. Vererbung am Centralnervensystem," Wien, 1907.

one group of five relatives. For some of these cases the spinal cord only was available. In the majority one or more of the relatives were fœtuses or new-born children; he had ten pairs of twins. He came to the conclusion that in the human species the inheritance of the convolutional pattern is a proved fact.

An excellent example was afforded by a series of three relative brains (mother, daughter and son), all of whom had the left hemisphere far less fissured and complete than the right hemisphere, although the three individuals were right-handed (Group XXVI). A peculiarity occurring in a relative brain on the left hemisphere will always, when inherited, occur on the same side in the other family members (gleichseitige Uebertragung, homologous transmission). Karplus here refers to conditions which are exceptions in the human brain, and which give low percentages as to their presence in the average pattern. (The peculiarities of which I have given statistics are those conditions which exist only in one hemisphere out of four in which the comparison was made.)

Karplus has made some measurements; he thinks individual variability covers to some extent the familial common type. He has also examined the medulla and the spinal cord in some pairs of relatives and has found family resemblances. Karplus then examined brains of *Macacus rhesus* (four pairs), of dogs (four groups of four, five, three, and three offsprings, and each time the mother), of cats (four groups of four, four, three and four kittens, and the mother cat for each of the groups) of goats (two she-goats and their twins, and a third pair of twins). He found that the family resemblance was *less* in the ape than in the dog and cat families. In the cat and dog the familial type and the peculiarities were inherited to some extent. In the macacus there was such a variability in the arrangement of the sulci that no definite similarity could possibly be noticed.

"The macacus brain shows a great variability of its sulci; some parts of the brain have a greater variability than others (occipital pole); undoubtedly the varieties of the sulci show a very great and striking identity in the two hemispheres of the same brain, but on the other hand, only for one single definite variety could a similarity between mother and offspring be observed" (pp. 71 and 160).

"The brain of the dog shows a great variability. The two hemispheres of the same brain are very similar. Peculiarities occur very often repeatedly in the same family. The two hemispheres of the same brain being very similar there is no occasion to find an homologous transmission" (p. 108).

From the brain of the cat the same conclusions were drawn as from the dog's brain (p. 137). In the goat's brain the variability was extreme, no similarity was to be found between the two hemispheres of the same brain, nor could any familial similarity be determined (Karplus reserves his opinion).

The human brain shows greater differentiation between left and right hemispheres than do the brain of the macacus, of the dog, and of the cat. The familial resemblance and the familial peculiarities are more numerous in the cat and in the dog than in the macacus. But the human brain shows the greatest familial resemblance, and the two hemispheres being differentiated, peculiarities may occur on one side only, and when transmitted by inheritance they will always reappear on the same side.

The macacus seems to be in the phylogenetic development of its zoological order, far behind the place occupied by the dog and the cat, which are at the summit of the carnivora. It would be interesting to examine less developed carnivora. Karplus thinks that it is possible to determine which are the more newly developed parts of the brain by the greater variability in their convolutional pattern. The same may be said for the spinal cord, where familial resemblance is less observed in the newer developed parts (pyramidal tracts) than in the older.

With the observations of Karplus as to the differentiation of left and right brain I fully agree, and I have given numbers to fix the opinions. With regard to the homologous transmission, I especially found this confirmed for the anastomosis of the transverse temporal with the superior temporal. The fact that inverted similarity only occurs in 1 per cent. of the cases also confirms the views of Karplus on the homologous transmission, although it shows that there may be exceptions. Neither Karplus nor myself have had the father and the mother examined with their child, and this would be necessary to make the demonstration complete. I have also demonstrated by many examples the possibility of determining by the degree or the index of variability which are the older parts of the brain. Also, for a sulcus, its similarity decreases in the latest developed parts. I demonstrated this in the case of the superior temporal, the posterior portion of which is the oldest and has twenty-seven for index of comparison, the middle portion five, the anterior portion three. On sixteen comparisons no complete differences (for the interruption) occurred in the posterior part, two occurred for the middle and three for the anterior part.

Juljusz Morawski examined cat and dog families and did not find

the same results as those quoted by Karplus. He found the most contradictory indications. No influence of sex could be traced. The brain weight had some influence on the advancement of the pattern; the body weight had no influence. Unilateral peculiarities were to be found in some cases. Morawski could not find any familial pattern transmitted by inheritance. The only positive fact he was enabled to observe was that some families showed a great number of peculiarities, other families no peculiarities at all.[1] This negative work need not cause any astonishment. Even if twenty observers had confirmed the findings of Karplus, a twenty-first would have been forthcoming to contradict them. This happens in the case of every scientific research and depends upon personal bias. In the present case, instead of being the twenty-first, Morawski was only the third. But he did his work in the laboratory of Karplus. His material was really too poor for the investigation of such a question, and it did not at all possess the value necessary for an experimental control. It would have been better had he examined grandparents, parents and offspring of one cat or one dog family, whereas, he always selected only the mother and the offspring. But this is not the time for discussing experimental methods, and I return to the discussion of human brains, the material of which, although not dependent on a selection, is proved to be of greater value and easier of examination.

Neither Karplus nor Schuster attempted to see whether it would be possible to discover, out of a group of brains, those which show the greatest resemblance and whether they belong to relatives. I cannot claim to have obtained that absolute proof of the familial resemblance, but I think that I have come nearer to obtaining it than my predecessors, as I have indicated the method of finding, out of a group of brains, that one which practically bears most resemblance to a given brain and therefore would have the greatest chance of being related to it.

Out of the series of thirty-two brains under my observation, the method which I had proposed one year ago showed for each brain its related companion of misfortune! Karplus had said:—

"It would be unreliable to give a numerical indication on the degree of similarity, as our knowledge of the variations of the sulci is still too incomplete, and as the statement of the authors on that subject are too much at variance" (p. 33).

[1] Morawski, Juljusz. "Gehirnuntersuchungen bei Katzen- und Hundefamilien," *Jahrb. f. Psych. u. Neur.*, Leipz. u. Wien, 1912, xxxiii, p. 306.

The trial of them which has been made in this paper shows, in my opinion, that arithmetical methods have already yielded such results as to indicate fresh lines for further research and to encourage and justify the prosecution of similar studies. The curves obtained for the answers to the principal questions show that their number is sufficient for the proposed examination. The regularity with which some averages, as, for instance, that of similarity, recur, is a proof of their constancy. Even with fifty-two questions alone, following the tables of Schuster, the figure 0·70 was obtained for the twin brains in my first paper. As a proof of accuracy I ask leave to recall that, having recorded 5,312 answers, I found out of them 968 similarities on the left hemispheres and 971 on the right hemispheres. The number of peculiarities on the left side, thus combining the conditions of the four hemispheres in each question, gave 223 and it was 225 for the right hemispheres.

In his most valuable studies on "The Law of Inheritance of the Mental and Moral characteristics in Man and its comparison with the Inheritance of the Physical Characters," Professor Karl Pearson arrives at a degree of resemblance of 0·5 for direct relationship. In another of his papers we find the following figures for the intellectual resemblance by sibship:—

Male and male	0·46	degree of resemblance.
Female and female	0·47	
Male and female	0·44	

In his "Grammar of Science" (1900), he had written:—

"The intensity of parental correlation is about 0·3 to 0·5, of grandparental about 0·15 to 0·3, and of fraternal about 0·4 to 0·6, the latter correlation being somewhat reduced when the "fraternity" consists of members of opposite sexes" (p. 459).

Such figures indicate the tangent of the angle which the line in correlation diagrams makes with the x axis. They are not to be compared with mine. Still, it is interesting to point out that the average of absolute similarity (+ + + +) between two relative brains was found to be 0·5, and that the absolute indexes of comparison gave:—

Male and male	9·42	index of comparison,
Female and female	9·49	
Male and female	8·06	

yielding thus final results approximating to those of Professor Pearson,

males being a little less similar than females and mixed comparisons giving the smallest figures.

The reason why I call the index of similarity an *apparent* index ought to be explained. The average of similarities amounting to 73 per cent., the remnant of 27 per cent. contains:—

(XI) incomplete similarities (as + − − +)	1, 9
(VI, VII, VIII, IX) peculiarities (as − + + +)	16, 9
(X) complete dissimilarities (as + − + −)	8, 2

Only the complete dissimilarities are to be taken as a term diminishing the value of the apparent similarities, as the peculiarities and the incomplete similarities contain potential possibilities of similarity, which heredity is capable of bringing about. It is a similarity for two hemispheres that the amount of their power of variability (which we may suppose to be correlated with adaptability) is the same; but we should be only able to measure it by the examination of a large series of brains belonging to one family, which is practically impossible at the present time. It is thus not possible to have a better estimation for an absolute index of comparison than to divide the similarities by the dissimilarities; the resulting figure shows how many times two brains more closely resemble than differ from one another. Next to that, the main attention must be concentrated on the number of conditions in which the two brains show a similarity of differentiation between left and right hemispheres.

The importance of the morphological inheritance of the sulci being proved, the significance of transitory forms becomes interesting. In many of the drawings the slow transition from one extreme condition to another is clearly to be followed up. This is especially the case in the lower end of the central sulcus—adjoining the lateral fissure or remaining remote from it—having an anterior or a posterior direction, and so on; in the connexions of the temporal transverse sulcus with the temporal superior and in the anastomoses of the ascending branches of the temporal sulci with the interparietal; in the different forms of the calcarine fissure, especially at its end, with the *play* of the sulcus lunatus. The significance of the deep gyri can also be traced as they become more superficial, when the brain mass or its complex fissuring augments. They may also be the result of the concealment of cortex when deeply compressed owing to the development of the neighbouring structures. The first method of origin gives the annectent gyri sectioning the inferior frontal sulcus, the interparietal sulcus and the

calcarine fissure. The second method concerns the cortex near to the sulcus diagonalis, the cortex between the two first temporal endings in the gyrus angularis, &c.

From some brains one derives the impression that an early condition of hydrocephalus has kept the sulci apart, preventing their anastomosis or making a normal extension inadequate to the intrinsic structure. Such an influence seems to be especially operative at the posterior end of the lateral fissure and its surroundings, near to the centre of the parietal bones, where the largest diameter exists in hydrocephalic skulls. It may cause the first temporal to be interrupted at its posterior portion. This occurred five times in my series, always confined to the left hemisphere, always in the children, four times in the bigger brains. The brain weight of the case in which it occurred in the smallest can be explained (XIV, B); it must have been higher during life; the brother's brain had a weight of 1,385 grm. It cannot be accurately estimated whether the female brain was more over the average than the male brain. The smallest of the brains showing that condition belonged to one out of six deaf and dumb children.

The cases are: III, B (deaf and dumb daughter); VII, B, adolescent insanity, daughter; VIII, B, son, early melancholia; XIV, B, sister, early mania; XVI, B, stillborn twin. The percentages of Retzius are 21 for the males (12 on the right, 27 on the left), and 4 for the females (8 on the right, 0 on the left). It diminishes the value of my explanation and makes further investigations necessary. Moreover, in the case of this interesting condition, the transitions are frequent and clinical notes do not supply details relating to the early years of the patients' lives nor as to the conditions in which they were born.

As for the general question of heredity, the convolutional pattern shows far more often slight transitions than suddenly appearing differences. Lamarck would find his theories confirmed and Weismann rather the reverse.

The knowledge of the different anastomoses and peculiarities of the sulci in the human brain is not yet based upon as great a number of investigations as the newer methods of biology would demand, and this is easily demonstrated by the comparison of the different numbers found by Cunningham, Retzius, Eberstaller and others. But a comparison with the statistics now at hand can nevertheless supply some indications of interest. Comparing the average figures of Retzius that yield the greatest differences with those of this study, we find that in all the cases, except in two, the figures resulting from the brains of our series indicate

a lack of annectent gyri and consequently *a greater number of anastomoses* of the sulci with each other. The normal brains described by Retzius had a heavier weight than that of ours, and it may be suggested that this factor is the main reason for the differences; but something more than this influences the figures. Differences relating to the parietalis superior may result from a different interpretation, especially when the parietalis superior is divided into two or more portions. What I considered as part of the *ps* joined with *pos*, may have been considered by others to be a mere secondary branch of *pos*. The same doubt arises in respect of the branches of the interparietal. The differences in the anastomoses of *ps* with the sulci præcunei gives less occasion for misinterpretations and confirm the opinion that heavier brains show a greater number of interruptions. The difference as regards question No. 74 arises from the manner in which the question has been put. Retzius asks for evidence of the existence of the rhinal sulcus, where I have thought that it is more important to look for its anastomosis with the lateral fissure. The concealment of the isthmus, no doubt being here possible, seems to be more frequent in series of lunatics, especially in males.

The statistics of Retzius relate to 100 hemispheres, seventy-five of which were of male and twenty-five of female brains. To make the comparison possible I have made the general percentage for Retzius's as well as for my cases, half of the male and the female percentages. Retzius's original general percentage in fact concerns an unequal mixture of male and female hemispheres. But I quite agree also that the newly given total averages may easily be subjected to criticism. All this proves how necessary it is to review these questions on the basis of a fresh array of carefully collected normal brains. Retzius makes interesting statements relating to the *sulcus interparietalis proprius* and shows that its connexions with the sulci postcentrales supply the following possibilities:—

TABLE K.—ANTERIOR CONNEXIONS OF THE INTERPARIETAL SULCUS.

Form of the connexion :	pos / poi ip					
Percentage of its existence in the Retzius series	55	17	11	9	4	4
Percentage of Cunningham	60	12	19	6·9	—	—
Percentage in my series	54	9	6	7	18	3
Notation	a	b	c	d	e	f

If we assign the initials a, b, c, d, e, f, to each of the modalities, and first write the initial corresponding to the left hemisphere of a brain, and then that corresponding to the right side, each brain will have its formula. The following table shows the inheritance of the different types :—

TABLE L.—DIRECT DESCENDING HEREDITY.

	I	II	III	IV	V	VI	VII	VIII	IX
Father	af	ee	aa	—	—	—	—	—	—
Mother	—	—	—	eb	ee	ae	aa	aa	aa
Son	ee	—	—	—	—	—	—	ca	bd
Daughter	—	ad	*da*	ea	aa	aa	*ab*	—	—

TABLE M.—DIRECT COLLATERAL HEREDITY (SIBSHIP).

	X	XI	XII	XIII	XIV	XV	XVI
Brother	fa	ae	—	—	ba	—	ea
Sister	—	—	aa	aa	*ca*	db	—,
Brother	ea	aa	—	—	—	bd	*cc*
Sister	—	—	aa	aa	—	—	—

The older brain has been put first. The formulæ in italics belong to the brains showing the posterior interruption of the superior temporal. The most common type seems to be *a*, eleven brains out of thirty-two have the formula *aa*; nine of them are females. The next common formula in the normal series shows a continuous post-central sulcus without connexion with the association area of the interparietal. But in the series of the lunatics, the next figure relates to the connexion of the postcentralis superior with the interparietalis and an independent postcentralis inferior.

Karplus's law of the homologous transmission is confirmed by the brain pairs No. III, IV, VI, VII, VIII, X, XI, XIV, and nothing invalidates it in the descending parentage. There is one case showing discrepancy (XV) by collateral parentage, but as we have not the pattern of both the parents, it is not possible to check the origin of the discrepancy.

The five cases showing a posterior interruption of the superior temporal have also a less anastomosed pattern than that of their parents or of siblings, except for XIV, A, where it also appears although differently interrupted in the elder brother, who has the type b.

Similarity of the type occurs in five pairs of hemispheres of the first group and in seven of the second group. Regression to the common

type of the population exists for six hemispheres of children, and deviation from the common type for seven hemispheres out of eighteen.

Further examples could be demonstrated to show the value of the hereditary resemblance in the study of the convolutional pattern. Hundreds of non-relative brains are needed to show that all kinds of variations exist in the infoldings of the human cortex, but with a series of relative brains it may readily be demonstrated that one form can slowly become transformed into another, and often in the four hemispheres of one pair of relative brains the slight transition from one extreme pattern to another extreme is clearly apparent.

It is a general conclusion arrived at in this paper that the convolutional pattern has, in itself, a morphological value which justifies a specialized research and makes it necessary that it should be examined first of all apart from any other structural condition. It is only when that study has been carried out exhaustively on a greater number of brain series, that we may venture to examine the reciprocal influence of other conditions upon the fissuration of the cortex. I allude, by the way, to the investigations of hereditary similarities and dissimilarities in the extension of the architectonic fields, which have to be carried out on an equal and as extensive as possible number of brains, before any comparative conclusions will be available.

(VII) Summary and General Conclusions.

Fifteen pairs of adult brains related by direct parentage, and one pair of twin brains have been examined according to a morphological and a statistical method, described in a previous paper of the author, and published in the *Philosophical Transactions of the Royal Society* (1916).

Eighty-three questions dealing with the convolutional pattern were answered in the case of each pair of brains, and the results summarized and divided according to their kind of similarity and dissimilarity.

A control of comparisons with non-relative brains enabled the author to demonstrate that relative brains are more like one another than non-relative brains.

The test, carried out on a pair of negro brains, proved that racial influences do not play an important part in the results.

A series of comparisons according to the weight of the brain demon-

strate that heavy adult brains show less resemblance to the average pattern than the smaller brains; but the weight of the brain is not a dominant factor in the results, and has no individual value; its influence does not diminish the essential family resemblance.

Comparisons of mixed sexes show less similarity than do comparisons between brains of the same sex, and the tables enable one to trace the origin of this diminution in resemblance in respect of the details of the brain pattern; hence an attempt has been made to indicate the main characteristics of the typical male and the typical female brain. These indications do not permit the author to assert that a given brain belongs to a male or to a female human being; they bear only a general character.

In the case of the brains of Hebrew brothers and in those of the Negroes, there were fewer variations in the pattern than in the brains of most of the British subjects, the racial origin of which were in all probability more mixed in type.

There are proofs of differentiation between left and right hemisphere, and the comparison of the hemispheres in the same brain with each other is worthy of careful attention; the amount of variations remaining nearly the same, an increase of differentiated conditions in one part of the brain is an indication of a greater resemblance in other parts.

The same method which has been used for the whole brain comparison has been used for each of the questions to be solved regarding the convolutional pattern, and it enables one to determine which part of the cortex is of more recent development in the ontogeny (which is proved by the comparison of fœtal brains), and also in the phylogeny (as suggested by comparative anatomy.

The study of relative brains is of special value in the determination of the transitions from one brain pattern to another, both as a whole and in detail. A knowledge of the normal transitions from one convolutional pattern to another may be of use in the consideration of the convolutional pattern in pathological conditions.

The definite results arrived at in this paper are summarized in the following conclusions:—

(1) The family resemblance of the convolutional pattern in man has an individual value. The family inheritance has a greater effect on the convolutional pattern than is exercised by the influence of brain weight or by sexual or racial influences.

(2) The apparent similarity between left and right hemispheres in the thirty-two human brains above described, and with the method indicated, is ... 0·73

The apparent similarity between homologous hemispheres, and thus also between the brains, of two persons of direct relationship, is ... ... 0·73

Thus one of the hemispheres of a human brain resembles the other hemisphere as much as it resembles the homologous hemisphere of a parent, a child or a sibling.

(3) The apparent similarity by direct relationship in man being ... ... 0·73

Incomplete similarities amount to ... ... 0·01,9

Peculiarities existing in one hemisphere out of four amount to ... ... 0·16,9

Complete dissimilarities amount to ... ... 0·08,2

1·00,0

(4) In ninety comparisons of non-relatives, the figures are:—

Apparent similarity	0·60,38
Incomplete similarities	0·02,923
Peculiarities	0·19,50
Complete dissimilarities	0·17,197
	1·00,0

(5) Considering that there is a constant resemblance shown for the hemispheres in man, we may believe that the apparent differences between the hemispheres of one brain consist of an amount of fixed and inherited differences and an amount of variable conditions, each of which can be revealed by the examination of brains related to the brain in question.

(6) In relative brains, the transition from one extreme pattern to another is imperceptible; there is continuity in the evolution.

Section of Psychiatry.

President—Dr. R. Percy Smith.

(*March* 13, 1917.)

Histological Examination of the Ovaries in Mental Disease.

By the late Laura Forster, L.R.C.P. & S.Edin., M.D.Berne.[1]

(Communicated by F. W. Mott, Major R.A.M.C.(T.), M.D., F.R.S.)

The following paper is the result of an investigation of human ovaries, carried out in the Pathological Laboratory, Claybury Asylum. The work was suggested to me by Dr. F. W. Mott, who considered that a systematic examination of the ovaries in cases of mental disease would be of medical and social importance.

There are two periods of life in the female, when the onset of insanity especially occurs—viz., early adolescence and the involutional period. This has been very clearly shown by Dr. Mott in a collective investigation of over 4,000 related cases, who are at present in, or who have been discharged from, or who have died in the London County Asylums. The parents of these insane offspring, in nearly 50 per cent. of the cases affected, have their first attack during the involution period from 45-60. Furthermore, Dr. Mott has found certain types of mental degeneration associated with absence of ova in the glands; others with absence of signs of maturation in the ova, both indications of sterility; he considers the latter is further evidence of "anticipation," by which the children of insane parents are affected at a much earlier

[1] From the Pathological Laboratory of the London County Council Asylums.

age than the parents, and in a more intense form. This may be a way in which nature tends to end, if it cannot mend, a degenerate stock.

As evidence of the correlation of brain and sexual glands, Ceni's experiments on birds are important. He removed one hemisphere, and the birds surviving the traumatic shock were killed after periods varying from a few months to three years, and their ovaries were subsequently examined histologically. The primary shock had the effect of causing them to cease laying eggs for some months. In the following year they began to lay again, but in the second year few eggs were laid, or they ceased altogether from laying. The birds were otherwise in a healthy condition. The examination of the ovaries showed premature progressive involution. The writer considers that the changes in the ovaries are an expression of a permanent state of functional torpor of the organ, in consequence of which ovulation is more limited than under normal conditions, and finally becomes entirely arrested. The arrest of this function causes a rapid and progressive involution, which affects the whole parenchyma of the ovary, and may be taken as a premature dynamic exhaustion of the sexual gland. The conclusions drawn are that there are very intimate dynamic relations between the brain and the ovary, the function of the latter being directly dependent on the anatomical and functional integrity of the brain. The writer considers that these facts have an important bearing on the question of reproduction in the physically unfit.

The ovary has of late received rather a prominent place as an important secreting gland. I refer my readers to Biedl's exhaustive work on internal secretion, where a very full list may be found of all the writers who have studied this question anatomically and physiologically. The parts of the ovary credited with the function of secretion are the corpus luteum and the atretic follicle. Limon made a comparative histological study of the ovaries of various animals, and states that the tissue arising out of the atretic follicle, through proliferation of the theca interna, consists of cells of epithelioid form, and he gives it the name of "glande interstitielle de l'ovaire. He has found these epithelioid cells in numerous groups imbedded in the stroma of certain animals. Fraenkel likewise made a detailed examination of the ovaries of animals, and also of human ones. He found these epithelioid cells in numbers in the ovaries of certain animals, more especially the rodents, but they were entirely absent in the anthropoids and man. He

concludes, therefore, that this tissue cannot be responsible for a large, general and important function.

Dr. Mott kindly supplied me with all the material available at the Pathological Laboratory, Claybury, and also sent me some from Charing Cross Hospital. For the rest, I am indebted to Dr. Turnbull, Director of the Pathological Institute, London Hospital, and to the following Medical Superintendents of the London County Asylums: Dr. Ogilvy, of Long Grove; Dr. Baily, of Hanwell; Dr. Gilfillan, of Colney Hatch; Dr. Stansfield, of Bexley; Dr. Lord, of Horton; Dr. Donaldson, of the Manor Asylum; Sir James Moody, of Canehill; and to Dr. Elkins, of Leavesden; and Dr. Campbell, of Caterham.

The ovaries were taken from 100 cases of persons who had died in the London County Asylums, and included cases diagnosed as dementia præcox, mania, melancholia, general paralysis of the insane, epilepsy and imbecility. To control the above cases, ovaries were taken from women and children who had died in the London and Charing Cross Hospitals. The control ovaries cannot, however, be considered as entirely satisfactory, as many of them were taken from persons who had died from a long-standing disease. For instance, those belonging to a woman, aged 32, with chronic interstitial nephritis, showed complete involution.

Methods.

The ovaries were fixed in Müller's fluid, and then taken through various strengths of alcohol. A certain number were embedded in paraffin, but for the majority celloidin was used, as it causes less shrinkage. The blocks were cut into serial sections, and in cases where the Graafian follicles were scanty in number, the whole of the ovary was cut through in this manner. In other cases it was considered sufficient to cut a series of one block only. The majority of sections were stained after cutting with hæmatoxylin and eosin. A certain number of blocks were stained by the alum-carmine method before cutting, and afterwards counter-stained with eosin.

Histology of the Normal Human Ovary.

The stroma of the human ovary consists essentially of a dense network of fibrous bands, interspersed in places by smooth muscle-fibres, and through which blood-vessels run. In the stroma are embedded the Graafian follicles (figs. 4 and 5, pp. 80, 81), each of which consists of

a layer of epithelium, bounded on its outer edge by a basement membrane. Lying within the follicle is the ovum, surrounded by its zona pellucida. As the follicle increases in size the epithelial layers become multiplied, and at a certain stage of development the discus proligerus is formed. This is a thickening of the epithelium at one end of the follicle, in which the ovum can be seen embedded (fig. 8, p. 82). The Graafian follicles vary considerably in number according to the age of the individual. In the new-born and quite young person the larger part of the stroma consists of follicles, but as age advances, and the follicles diminish in number, either by rupture or atresia, their place is filled by the so-called corpora lutea, which, in their turn disappearing, give way to fibrous tissue. I have not observed in the human ovary those groups of epithelioid cells noticed by Heape in abundance in the rabbit, and by Limon in the rabbit and rodents. I noticed two principal forms of corpus luteum which occur in varying numbers throughout the stroma, the size of which varies considerably. The first I have not been able to follow in its development, and have only noticed it in one phase, where it consists of a nodule of non-cellular protoplasm (which takes a deep eosin stain) separated off into columns by thin trabeculæ of connective tissue, which converge to a central strand, forming the axis of the nodule. I believe this is the form which follows the rupture of a follicle (fig. 6, p. 81). The second form is the one taking the place of the follicle which undergoes atresia. It consists of a nodule of loosely arranged connective tissue, separated off by delicate bands of denser connective tissue. I have noticed a number of these in process of formation round a degenerating ovum which has lost its surrounding membrana granulosa (figs. 1, 2 and 3, pp. 79, 80). The zona pellucida often remains intact after the nucleus has lost its power of staining, and the plasma is shrunken.

These two forms of corpus luteum are quite different in appearance, and according to Paladin and Beigel's acceptation of the terms, I have called the former "corpus luteum verum," and the latter "corpus luteum spurium." The name of corpus luteum verum is attached by some authors to that form only which occurs when pregnancy takes place. However, as the difference between those occurring with or without pregnancy seems to be only a question of size, the designation does not seem to me justified, and as Lawson Tait says, "the belief that there is such a thing as a true corpus luteum in relation to pregnancy is one of the most extraordinary crazes that has crept into medical belief."

Results of the Histological Examination.

As it was impossible in so large a number of cases to estimate the absolute number of follicles present in each ovary, I adopted as a method of comparison the plan of counting the largest number present at any one level throughout the sections examined. I append here a tabulated statement of each case, with the results summarized, and give below a summary of comparative results according to age and mental disease.

Dementia Præcox.—In this group the ovaries of all those who had reached the age of 30 showed signs of early involution, marked by an increase of interstitial connective tissue, and a great scarcity of Graafian follicles. Even in those much below this age, there was a distinct diminution of the follicles, as compared with a normal woman of the same age. As an instance, the greatest number of follicles at one level, found in one case aged 19, at death, was fifty-nine, whereas in a control ovary from a girl aged 24, there were 171 follicles at one level; and in another aged 17, there were 454 counted at one level.

Imbecility with or without Epilepsy.—These ovaries did not present a uniform type. Some quite young persons showed early involution, while in others there were a proportionately large number of follicles. In one case, for instance, of congenital imbecility with epilepsy, aged 39 at death, 123 follicles were found at one level, and no true corpora lutea.

Melancholia.—(Many of these cases are probably mania depressiva, as true melancholia is met with at the involutional period.) Here we find a definite diminution of follicles. The greatest number at any one level was forty, in the ovary of a woman aged 23,[1] and a number of these were undergoing cystic degeneration, and showed no trace of an ovum. In the majority of cases, the numbers found at one level ranged from three to fifteen, and the stroma showed a marked increase of fibrous tissue, and some thickening of the walls of the blood-vessels.

Mania.—In all these cases a marked diminution of follicles was found, the largest number at any one level being thirty-two, in a woman aged 32.

General Paralysis.—Here there was likewise a decrease in the number of follicles, the greatest number in any one case at any one

[1] It is probable this was a case of dementia præcox.—F. W. M.

level amounting to forty, with the exception of a case of juvenile general paralysis, aged 20, where the largest number amounted to 160 at one level. There was a marked increase of fibrous tissue, and thickening of the walls of the blood-vessels, some of which were almost obliterated.

Conclusions.

It would appear from the above results, that where there is disease of the brain, or mental incapacity associated with it, the power of the individual to reproduce her kind, if not absolutely cut off, is at least diminished, and in most cases an early cessation of the ovarian functions seems to take place. These facts argue that there is an intimate relation between the ovary and the brain, and confirm Ceni's statement on this point.

I do not in this paper intend to discuss in full as to whether the ovary plays a direct part in the internal secretion of the body, or whether its influence is only an indirect one. With regard to its histology, however, I can say that there is no part in the structure of the human ovary that leads me to suppose it can exercise a secreting function, unless it be the Graafian follicle itself. Neither the corpus luteum verum, nor the corpus luteum spurium, show in their composition anything resembling epithelial cells, and it is these two bodies, more especially the atretic follicle, which in the lower animals are credited with the function of secretion. After a careful study of all the ovaries, normal or otherwise, that come under my notice, I can only confirm Fraenkel's statement, that in the human ovary there are no groups of epithelioid cells corresponding to those found in the rabbit and other animals, and representing the "glande interstitielle" of Limon.

My thanks are due to the London County Council Asylums Committee for kind permission to work in the Laboratory, to Dr. Mott for his suggestions and help, and to Mr. Charles Geary for kindly taking the photographs which illustrate this paper.

TABLE I.

No.	Nature of disease	Age at first attack	Age at admission	Age at death	Condition of ovaries	Menstruation	Pregnancies and results	Married or Single
1	Dementia præcox	19	19	24	Connective tissue increased; 11 ova at one level; some corpora lutea present	Regular until one year before death	*Nil*	Single
2	Dementia præcox	29	30	33	38 follicles at one level; some corpora lutea present	Began at 13; ceased two years before death	*Nil*	Single
3	Stupor and rapid mental enfeeblement	39	39	39	Advanced involution; no follicles detected; no corpora lutea seen	Began at 14 and ceased seven months before death	*Nil*	Married
4	Dementia præcox; delusional insanity	19	19	19	59 follicles at one level; no true corpora lutea noticed	No history	*Nil*	Single
5	Suicidal dementia	24	32	34	35 follicles at one level and mostly immature ones; a few small corpora lutea vera present	Began at 16; irregular during mental attacks	*Nil*	Single
6	Chronic mania; dementia præcox; progressive deterioration	29	29	32	Marked increase of connective tissue; 18 follicles at one level; all immature, corpora lutea vera present	Ceased a few months before death	*Nil*	Single
7	Delusional insanity	33	34	36	Excess of connective tissue; 6 follicles at one level; corpora lutea vera present	Menorrhagia for thirteen years, regular up to time of last illness	Three children, none living	Married twice
8	Primary dementia; katatonia; dementia præcox	28	29	30	Connective tissue increased, only 3 follicles at one level	Began at 14; irregular; ceased twelve months prior to admission	*Nil*	Single
9	Katatonia	30	30	37	Excess of connective tissue, only 2 follicles at one level	No history	Three children, last one born 6 weeks prior to admission	Married
10	Dementia præcox	25	25	30	Great increase of connective tissue; 12 follicles at one level; a few corpora lutea vera present	Regular	*Nil*	Single
11	Dementia præcox	17	19	26	25 follicles at one level; corpora lutea vera present	Normal during detention	*Nil*	Single
12	Dementia with increasing stupor	25	25	29	32 follicles at one level; some small corpora lutea vera present	Very irregular	*Nil*	Single

No.	Nature of disease	Age at first attack	Age at admission	Age at death	Condition of ovaries	Menstruation	Pregnancies and results	Married or Single
13	Dementia præcox	23	25	29	16 follicles at one level; small corpora lutea vera present	Began at 18, ceased just before death	*Nil*	Single
14	Dementia with increasing stupor	29	36	41	3 follicles at one level; some increase of connective tissue; corpora lutea vera present	Did not occur during detention	*Nil*	Single
15	Dementia præcox	28	29	32	Connective tissue increased; 3 follicles at one level	No history	*Nil*	Single
16	Dementia præcox	24	26	30	6 follicles at one level; connective tissue increased; corpora lutea vera present	Occurred at irregular intervals during detention	One child living	Married
17	Dementia præcox	26	26	27	25 follicles at one level; connective tissue somewhat increased; corpora lutea vera present	Regular up to admission; did not occur during detention	One child, 4 years old, living	Married
18	Paranoid dementia, præcox	25	25	34	6 follicles at one level; small corpora lutea vera present	Dysmenorrhœa during detention; ? previous history	*Nil*	Married
19	Dementia præcox	26	26	32	8 follicles at one level; connective tissue increased; corpora lutea vera present	Normal during detention	*Nil*	Married
20	Stuporose insanity	34	34	35	No follicles detected	Began at 13; history of regularity	Two miscarriages, two stillborn, none living	Married thirteen years
21	Confusional insanity	34	34	34	10 follicles at one level; excess of connective tissue; a good many corpora lutea vera present	Regular until three years before admission	*Nil*	Widow, married for three years
22	Epilepsy with dementia	18	31	36	108 follicles at one level; a few small corpora lutea vera present	Free and regular; last time fourteen days before death	No history, but there are abdominal striæ present	Single
23	Cretin	Congenital	28	29	13 follicles at one level	Irregular	*Nil*	Single
24	Congenital imbecility minus epilepsy	Congenital	18	25	13 follicles at one level; connective tissue increased; corpora lutea vera present	Never occurred	*Nil*	Single
25	Imbecile with congenital epilepsy	Congenital	32	37	5 follicles at one level; connective tissue increased; large corpora lutea vera present	Regular till about three months before death	*Nil*	Single

26	Congenital imbecility minus epilepsy	Congenital	30	33	51 follicles at one level	Regular	*Nil*	Single
27	Congenital idiocy with epilepsy	Congenital	15	18	13 follicles at one level; no true corpora lutea noticed	Never occurred	*Nil*	Single
28	Congenital idiocy with epilepsy	Congenital	22	22	12 follicles at one level	Never occurred	*Nil*	Single
29	Epileptic imbecile	Congenital	35	38	Only a few isolated follicles present; numerous corpora lutea vera	Irregular	Abdominal striæ present; no history	Single
30	Insanity with epilepsy	34	34	36	17 follicles at one level; some increase of connective tissue; a few corpora lutea vera present	Regular, menorrhagia	*Nil*	Single
31	Epileptic	12	23	24	204 follicles at one level	Regular	One child	Married 5½ years
32	Epileptic idiot	—	24	28	43 follicles at one level; true corpora lutea present	Regular; occurred three weeks before death	*Nil*	Single
33	Imbecility with epilepsy	Probably congenital	15½	23	4 follicles at one level; connective tissue increased; small corpora lutea vera present, but the spuria predominate	No date of onset; menorrhagia during detention	*Nil*	Single
34	Imbecility with epilepsy	Congenital	18	24	25 follicles at one level; connective tissue increased	Normal; date of onset not known	One child	Married
35	Imbecile	Congenital	24	24	96 follicles at one level; corpora lutea vera present	Amenorrhœa; ? past history	*Nil*	Single
36	Imbecility with epilepsy	Probably congenital	18	21	45 follicles at one level; no true corpora lutea	Began at 19, ceased shortly before death; appears to have been regular	*Nil*	Single
37	Imbecile	—	24	24	6 follicles at one level; corpora lutea vera present, but the spuria exceed them in numbers	No history	*Nil*	Single
38	Epilepsy; feeble-minded	Six weeks old	15	22	6 follicles at one level; no true corpora lutea vera present	Very irregular	*Nil*	Single
39	Idiocy with epilepsy	Congenital	34	39	123 follicles at one level; a few small corpora lutea vera present	Never occurred	*Nil*	Single
40	Idiocy with epilepsy	Congenital	15	19	3 follicles at one level; considerable increase of connective tissue; no corpora lutea vera	Irregular	*Nil*	Single
41	Insanity with epilepsy	22	22	27	31 follicles at one level	Profuse dysmenorrhœa	*Nil*	Single
42	Stupor; ? dementia præcox	17	17	33	Very few follicles detected; connective tissue increased	Regular	*Nil*	Single
43	Intelligence of low order; dull and apathetic; epileptic, imbecile	2	14½	17½	14 follicles counted at one level, some dilated; corpora lutea vera present, but the spuria predominate	Ceased three years prior to admission	*Nil*	Single

No.	Nature of disease	Age at first attack	Age at admission	Age at death	Condition of ovaries	Menstruation	Pregnancies and results	Married or Single
44	Insanity with epilepsy; typical insane epileptic temperament	21	23	26	500 follicles at one level, also a good many cystic dilatations of follicles; corpora lutea vera present	Began at 18, usually irregular	*Nil*	Single
45	Imbecile	Congenital	28	29	Only three follicles at one level: considerable increase of connective tissue; Corpora lutea vera present	Regular; began at 17	*Nil*	Single
46	Very slight intelligence	Congenital	18	21	36 follicles at one level; no true corpora lutea	Did not occur during detention	*Nil*	Single
47	Low type of imbecile	Congenital	15½	25	15 follicles at one level; corpora lutea vera present	Began at 15	*Nil*	Single
48	Organic brain disease, imbecile	16	16	18	Crowds of small follicles, also dilated ones; no definite corpora lutea noticed	Never occurred	*Nil*	Single
49	Idiocy with epilepsy	Congenital	15	19	41 follicles at one level, some quite dilated with healthy-looking ova	Never occurred	*Nil*	Single
50	Congenital idiocy with epilepsy	Congenital	17	25	3 follicles at one level	Occurred about every three months	*Nil*	Single
51	Congenital idiocy minus epilepsy	Congenital	25	29	7 follicles at one level; corpora lutea vera present	Never occurred	*Nil*	Single
52	Imbecile	Congenital	18	25	274 follicles at one level; some small corpora lutea vera present, but the spuria predominate	Very irregular; ceased some time before death	*Nil*	Single
53	Insanity with epilepsy	19	35	41	2 immature follicles only; advanced involution; a few small corpora lutea vera present	Very irregular	*Nil*	Single
54	Imbecile	Congenital	22	31	42 follicles at one level; a few small corpora lutea vera	Began at 10 years	*Nil*	Single
55	Feeble-minded; memory defective; dull and apathetic	35	36	36	6 follicles at one level; corpora lutea vera present	Began at 13; continued regularly	2	Married
56	General paralysis of the insane	42	43	44	Marked increase of connective tissue; a few isolated follicles only	No history obtainable	15	Married twice
57	Organic brain disease	35	35	37	6 follicles at one level; corpora lutea vera present	Once during detention	1	Married six years

58	Progressive general paralysis	38	38	41	Involution advanced; very few follicles present	Regular	One child	Married
59	General paralysis	34	34	38	28 follicles at one level, mostly immature; numerous small corpora lutea vera	No history; absent during detention	Four children, all living	Married
60	General paralysis	43	43	43	11 follicles at one level; a good many corpora lutea vera	No history; absent during detention	No history; abdominal striæ present	Married
61	General paralysis, beginning with maniacal symptoms	38	40	41	Involution advanced, only one or two isolated follicles seen	No history obtainable	Several miscarriages	Married
62	General paralysis	36	36	38	4 follicles at one level; some large corpora lutea vera present	Twice during detention	4	Married fourteen years
63	General paralysis	36	38	43	Advanced involution, only 2 follicles noticed	Did not occur during detention	5	Married fifteen years
64	General paralysis	?	21	24	40 follicles at one level; corpora lutea vera present	Nothing known	*Nil*	Single
65	General paralysis of the insane	40	40	41	Involution advanced; 5 follicles at one level, mostly degenerating; numerous corpora lutea vera	Frequent and excessive	3; first and second miscarried, third died soon after birth	Widow
66	General paralysis	27	29	30	Only 2 follicles at one level	Regular; began at 15, ceased shortly before death	*Nil*	Single
67	General paralysis	26	26	26	23 follicles at one level; a few corpora lutea vera present, but the spuria predominate	Did not occur during detention	*Nil*	Single
68	General paralysis	35	35	36	Both ovaries consist of a narrow band of normal stroma enclosing a mass of corpus luteum tissue; in the normal tissue there are 42 follicles at one level	No history obtainable	Abdominal striæ present	Married
69	General paralysis	39	39	41	Involution almost complete	Occurred only once during detention; began at 15	?	Married one year
70	Juvenile general paralysis	19	19	20	160 follicles at one level; some large corpora lutea vera present	Regular until just before admission; began at 15	*Nil*	Single
71	General paralysis	43	43	44	Advanced involution; no follicles detected	No history obtainable	Thirteen children	Married
72	General paralysis, demented type	35	35	38	Involution advanced; 11 follicles at one level; corpora lutea vera present	Regular till eighteen months before admission	*Nil*	Single
73	General paralysis	37	38	39	34 follicles at one level, some dilated; corpora lutea vera present, but the spuria predominate	Ceased three years prior to admission	One child living	Widow

No.	Nature of disease	Age at first attack	Age at admission	Age at death	Condition of ovaries	Menstruation	Pregnancies and results	Married or Single
74	Melancholia	44	44	44	Only 3 follicles at one level	Occurred during detention	Six children living	Married
75	Melancholia; suicidal at an early stage	41	41	46	A few isolated follicles present; involution almost complete; a few corpora lutea vera present	Three times during detention	*Nil*	Married twenty-one years
76	Melancholia	39	39	39	Only two follicles at one level	Began at 16; ? regularity	*Nil*	Single
77	Chronic melancholia	38	41	44	5 follicles at one level; corpora lutea vera present	Regular during detention; occurred five days before death	History defective	Married
78	Melancholia	26	29	33	9 follicles at one level; marked increase of connective tissue	Began at 14; history of regularity	Three	Married
79	Melancholia	34	36	40	8 follicles at one level; excess of connective tissue; corpora lutea vera present	Began at 14; fairly regular	Seven; last child born six weeks prior to admission	Married
80	Melancholia agitata	27	27	27	3 follicles at one level; connective tissue increased; some small corpora lutea vera present	Did not occur during detention	*Nil*	Single
81	Melancholia	28	28	37	Involution almost complete; 3 follicles at one level; corpora lutea vera present	Regular	Seven; three children living	Married
82	Melancholia	24	34	39	Only 1 follicle noticed	Regular and profuse	*Nil*	—
83	Melancholia	35	36	36	4 follicles counted at one level; a few dilated ones; corpora lutea vera present	History of irregularity	One child living, 8 years old	Married
84	Puerperal mania, which became chronic	34	34	41	Advanced involution; 6 follicles at one level; some large corpora lutea vera present	Regular	Four; three children living	Married
85	Mania	41	42	44	Advanced involution; only a few isolated degenerated follicles found	No history	*Nil*	Married
86	Mania with epilepsy	?	27	28	Excess of connective tissue; 3 follicles at one level	Not regular; occurred three times during nineteen months' detention	*Nil*	Single

87	Mania with epilepsy	20	23	30	3 follicles at one level	Regular; occurred one month before death	*Nil*	Single
88	Mania with imbecility	?	22	32	Connective tissue increased; 32 follicles at one level; corpora lutea vera present	Dysmenorrhœa; ? commencement	*Nil*	Single
89	Mania with epilepsy	22	22	24	24 follicles at one level; no corpora lutea vera noticed	Ceased two months before admission	*Nil*	Single
90	Mania	33	33	33	6 follicles at one level; connective tissue increased; corpora lutea vera present	No history obtainable	One child	Married
91	Recurrent mania	29	29	29	5 follicles at one level; interstitial connective tissue increased	No history obtainable	*Nil*	Single
92	? Acute mania	21	21	21	8 follicles at one level; corpora lutea vera present	Regular	*Nil*	Single
93	Mania	?	16	40	3 follicles at one level; some large corpora lutea vera present	Irregular; scanty	*Nil*	Single
94	Acute post-puerperal mania	26	26	30	39 follicles at one level; connective tissue increased	Did not occur during detention	One child born three weeks prior to admission	Married
95	Died after recent pregnancy	24	24	24	5 follicles at one level; some small corpora lutea vera present	Regular	Two	Married
96	Consciousness clouded, confusion, hallucinations	22	26	34	10 follicles at one level; no dilated ones; corpora lutea vera present	*Nil* noted	Two; youngest thirteen months old	Married
97	Degenerate in mind and body	23	23	23	40 follicles at one level; connective tissue increased; mostly degenerated	Began at 16	*Nil*	Single
98	Melancholia	37	37	37	30 follicles at one level; connective tissue increased; several corpora lutea vera present	Once during detention	*Nil*	Single
99	Mania	22	23	34	3 follicles at one level; a few corpora lutea vera present	No history obtainable	*Nil*	Single
100	Epileptic	38	38	41	Involution advanced; very few follicles present; a few corpora lutea vera present	No history	*Nil*	Single
101	Epilepsy with mania	Two previous attacks	24	42	Involution advanced; very few follicles; a few corpora lutea vera present	No history	*Nil*	Single

TABLE II.—CONTROL OVARIES OF PERSONS NOT AFFECTED WITH MENTAL DISEASE.

No.	Age	Cause of death	Condition of ovaries
A	30	Corrosive acid poisoning	Graafian follicles not numerous, 9 at one level; connective tissue much increased; numerous corpora lutea vera present
B	33	Cirrhosis of liver	12 follicles at one level; a good many degenerate follicles which contain no ova; numerous corpora lutea vera; connective tissue increased
C	4	Extensive burning	Innumerable follicles present, some of which are much dilated; no corpora lutea of any description noted
D	4 days	Pneumonia	Innumerable follicles, a number of which are dilated; no corpora lutea of any description
E	25	? Poisoning	Follicles at all stages of development; 81 at one level
F	7 months	Catarrhal gastritis and enteritis	Innumerable follicles, many dilated ones; no corpora lutea of any description noticed
G	32	Heart failure; chronic interstitial nephritis	No ova detected; involution appears complete; numerous corpora lutea vera present
H	17	Heart failure; diabetes	454 follicles counted at one level, some of which are considerably dilated; a few corpora lutea vera and spuria present
I	35	Cerebral hæmorrhage; chronic interstitial nephritis	Very few follicles present, and these are mostly degenerating; increase of connective tissue and thickening of blood-vessel walls; many corpora lutea vera present
K	24	Diabetes	171 follicles at one level; connective tissue somewhat increased and walls of blood-vessels thickened; corpora lutea vera and spuria present
L	31	Carcinoma recti	78 follicles at one level; corpora lutea vera and spuria present
M	35	Chronic empyema	14 follicles counted at one level; corpora lutea vera present
N	28	Exophthalmic goitre	10 follicles at one level; corpora lutea vera present

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FIG. 1.

L. S., aged 25, congenital imbecile. Connective tissue proliferation round degenerating ovum, and formation of corpus luteum spurium. (× 85.)

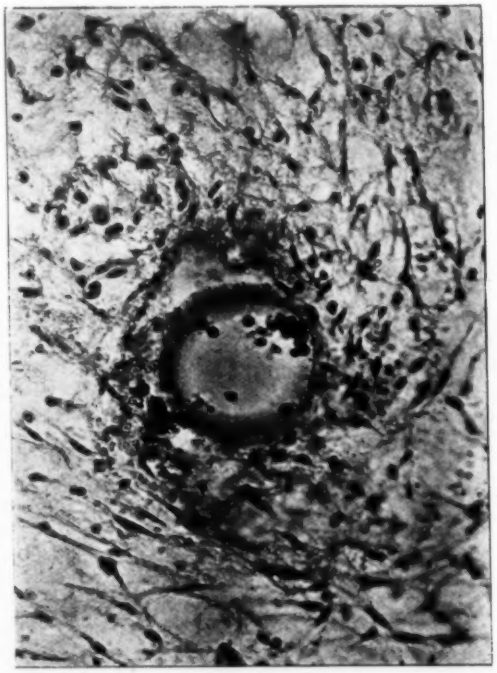

FIG. 2.

The same as fig. 1. (× 300.)

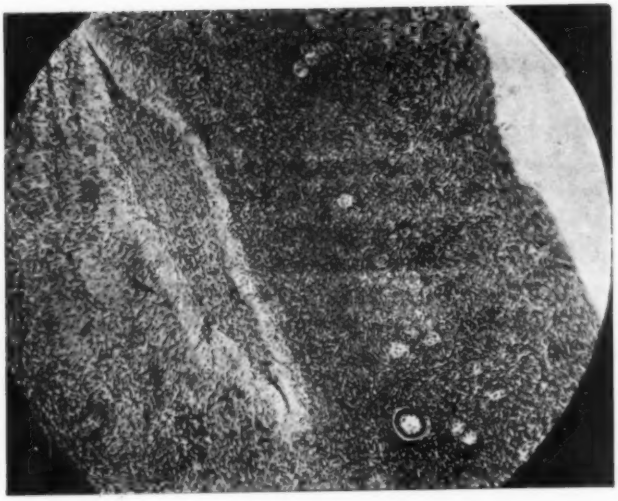

FIG. 4.

Control ovary, aged 24. Edge of ovary with Graafian follicles and corpus luteum spurium. (× 85.)

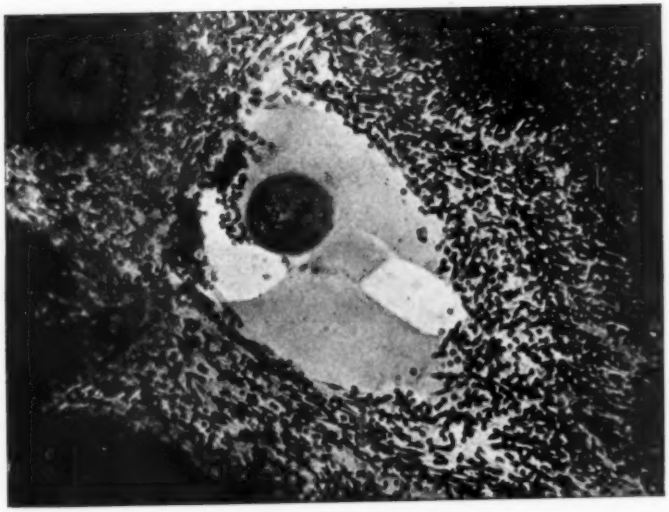

FIG. 3

L. E., dementia præcox, aged 23. Beginning of connective tissue proliferation round degenerating ovum. (× 180.)

FIG. 6.

E. W., aged 41, general paralysis of the insane. Corpus luteum verum surrounded by ovarian stroma. (× 85.)

FIG. 5.

Control ovary, aged 24. Edge of ovary with Graafian follicles. (× 85.)

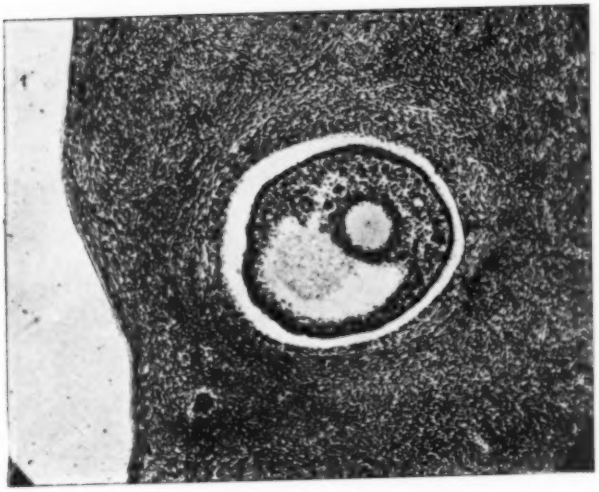

FIG. 8.

N. F., aged 39, general paralysis of the insane. Edge of ovary with Graafian follicle embedded in the stroma. (× 85.)

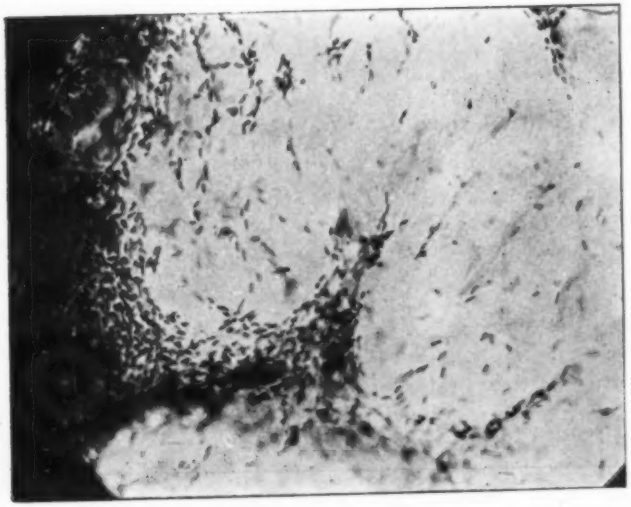

FIG. 7.

Control ovary, aged 33. Portion of corpus luteum verum. (× 200.)

ADDENDUM BY F. W. MOTT, Major R.A.M.C.(T.), M.D., F.R.S.

Before the outbreak of the War Dr. Laura Forster handed me the paper which has just been read. Unfortunately she immediately left England and I was unable to communicate with her. But for this I should have suggested some modifications and additional microscopic investigations of the sections which she has prepared with so much diligence. I must publish the paper as it was written, for her death has been recently announced, which is greatly to be regretted, as she was an earnest worker for whom I had great respect. I thought it desirable, however, from a study of the great number of sections made by Miss Forster to write a commentary, which, had she lived, I would have asked her to consider with a view to its partial or complete embodiment in her paper.

This investigation is beset with many difficulties, and I think that if fewer cases with perfect notes had been selected more convincing results would have been obtained. Still the large number of cases studied permits some generalizations to be made which a smaller number would not do. Among the difficulties which Miss Forster had to contend with was the possibility of variable diagnosis of the mental disease by the medical officers in charge of the cases. Again, her observations show that chronic diseases of long standing—e.g., nephritis, chronic endocarditis, and diabetic cirrhosis—may cause follicular degeneration and fibrotic atrophy. Lastly, it is difficult to make a complete histological examination of the ovary; for owing to its structure the paraffin method cannot be employed for serial sections with advantage, and it was found that the only satisfactory mode of procedure was to embed the material in celloidin. The blocks were cut and each section was laid out in series on glass plates, examined with a low power objective, and then, if a particular follicle was required to be studied, the series containing it was stained and mounted.

Nevertheless, taking into consideration the large number of ovaries thus carefully examined by Miss Forster, and fully admitting the difficulties of drawing certain conclusions owing to the complicating causes mentioned, I am of opinion that her work establishes the fact that there is a correlation between certain types of mental disease and morbid changes in the ovaries. I have not liked to add my own views to her communication without consulting her. I have, therefore, given it as she handed it to me. If it had been possible, I should have asked

her to more carefully consider the following points in relation to the morbid histology of the ovaries and their possible association with certain types of mental disease. The corpora lutea vera she very properly distinguishes from the corpora lutea spuria, or, as I should prefer to name them, the corpora atretica. The former are the result of the maturation and rupture of normal Graafian follicles, the latter are the result of invasion of a degenerate follicle by the stroma of the gland. It is permissible to assume that the existence of a large number of the former of large size is evidence that for some considerable time during the possible reproductive life of the woman healthy follicles were maturing and rupturing. Consequently a comparison of the relative numbers and size of the corpora lutea vera in relation to (*a*) the age of onset of the mental disease and age at death, associated with (*b*) history of menstruation when obtainable would have been valuable. Some idea might be gathered, therefore, when the normal ovarian function of follicular maturation ceased, and how far this could be connected with, or be considered coincident with, the onset and progress of the mental disease.

But suppose the follicles, instead of bursting, undergo degeneration with their contained ova and form the corpora atretica, we then have evidence of a morbid condition, consequently the relative numbers of the corpora atretica to (*a*) corpora lutea vera, (*b*) the age of onset of the mental disease and age at death, would afford some indication when normal follicular maturation ceased (if it ever occurred), and how far the appearance of the degenerative follicle could be connected with, or considered coincident with, the onset and progress of the mental disease. The examination of a large number of the sections prepared by Miss Forster has impressed me with two facts, viz., that there is evidence to show that the normal maturation of the follicle and formation of the corpus luteum verum tends to cease and be replaced by the degeneration of the follicle with the onset of certain types of mental disease. I will, therefore, consider a little more fully the characteristics which serve to distinguish the degenerate or atretic follicle from the true corpus luteum, and subsequently I will refer to the particular cases which illustrate the premise I have stated.

(1) The atretic or degenerate follicle exhibits no indication of rupture to the exterior.

(2) The follicular epithelium forming the zona granulosa and discus proligerus instead of hypertrophying degenerates, separation from the wall and fragmentation occurs, the chromatin substance instead of

appearing as a distinct nucleus is seen as fine points in the cytoplasm, much smaller than the nucleus. They lose their basophil staining reaction, break up eventually and become unrecognizable as cells, finally disappearing altogether. Hæmorrhage into the degenerate follicles is very liable to occur.

(3) The ovum is retained in the follicle but undergoes degenerative changes, it may lose its regular circular shape, the zona pellucida persisting. The germinal vessels and germinal spot lose their circular form and have an irregular appearance; the ovum may shrink away from the zona pellucida and finally it may be invaded by cells.

(4) The connective tissue wall does not proliferate to form a network among the epithelial cells as occurs in the true corpora lutea, and there is usually no ingrowth from the theca until the epithelial cells are in an advanced stage of degeneration or have altogether disappeared.

Such degenerative changes may occur in all stages in the development of the follicle, and not merely in the fully formed follicle that has failed to rupture. Atretic follicles may continue for a time as cysts which remain filled with fluid.

Heape has shown that in the rabbit two kinds of degeneration prevail: in the one kind the changes first affect the follicle then the ovum; in the other the ovum is first affected and the follicle afterwards. Heape interprets the latter change as evidence that the ovum is not capable of assimilating the nourishment brought to it. The more usual cause of degeneration in immature follicles is lack of sufficient nutriment or of nutriment of the requisite kind. (Marshall, "Physiology of Reproduction.")

I have examined sections from the following cases on the lines indicated :—

(1) A cretin, S. B., aged 28 on admission to Leavesden; died at the age of 29. No corpora lutea vera seen; atretic follicles, a few immature follicles, and dense fibrous tissue. In the left ovary, in addition, the organ is the seat of a large blood cyst.

(2) A. B., aged 18 on admission to Leavesden; age at death, 21. Never menstruated. No sign of mature Graafian follicles, a few atretic follicles, very few small corpora lutea, paucity of immature ova.

(3) M. T., admitted to Leavesden, at the age of 18, suffering with idiocy and epilepsy; never menstruated; died at the age of 22. Excessive fibrosis; many atretic follicles in various stages. A Graafian follicle of small size with discus proligerus, and ovum with zona pellucida observed. The ovum shows

the first stage of degeneration. There is a vacuolation in the place of the germinal vessels and germinal spot.

(4) A. S. P., aged 23 at death. Dementia præcox. Died of tuberculosis. Interstitial connective tissue much increased. Very few immature follicles. Atretic follicles, hæmorrhage into one of them. One ovum in a dilated follicle seems to be dividing into segments; the zona pellucida appears normal. There is a hæmorrhage into the cavity of the follicle; it no longer contains any epithelial lining cells. Corpora lutea vera, a few present.

(5) E. M. S., aged 30 at death. Post-puerperal mania. There has been some chronic inflammatory process, for the ovarian tissue seems quite encapsuled in connective tissue. A small Graafian follicle seen with normal ovum; discus proligerus and zona granulosa.

(6) B. W., aged 33 at death from cirrhosis of liver and ascites. Charing Cross Hospital. The ovaries are large and weigh respectively: Right, 13·9 grm.; left, 20·2 grm. The increase in size is due to a number of degenerate follicles of varying size; there are also a number of corpora lutea vera. There is an increase of dense fibrous tissue, which, by its contraction around the cysts, gives a nodular appearance to sections of the gland. There are only a very few immature follicles. I have seen several ovaries of chronic drunkards presenting this appearance. The corpora lutea vera may be regarded as evidence of normal follicular maturation in earlier life before the effect of the alcohol had produced the morbid change and degeneration.

(7) E. F., admitted to Bexley for melancholia at the age of 29; died aged 33. Few corpora lutea vera; great increase of connective tissue; many atretic follicles.

(8) A. M., admitted to Bexley at the age of 22; died at the age of 31. Right ovary, 16 grm.; left ovary, 16 grm. The large size of the ovaries is due to corpora lutea vera in fair numbers, increase of connective tissue, and a large number of atretic follicles.

(9) L. M. S., admitted to Bexley at the age of 25 for dementia præcox; died at the age of 30; right ovary, 3·7 grm.; left ovary, 3·7 grm. The section shows fibrotic atrophy in a remarkable manner. There are only a few scattered immature follicles. Atretic follicles are seen in all stages of degeneration. There are hardly any corpora lutea vera present in either ovary. There is a great increase of connective tissue and some thickening of the vessels.

(10) E. T., aged 19, admitted to Long Grove with juvenile general paralysis; died at the age of 20. Menstruation ceased prior to admission; previously regular. Crowds of follicles in both ovaries. A few rather large corpora lutea. A number of atretic follicles.

(11) O. H., admitted to Horton at the age of 19 for dementia præcox; died at the age of 26. Right ovary, 14 grm.; left ovary, 9·5 grm. Although the

ovaries are large there are only a few small corpora lutea. The glands are very vascular and the vessels are dilated and congested. There are numbers of atretic follicles in all stages of degeneration and subsequent fibrotic change.

(12) L. L. N., admitted to Manor Asylum. Diagnosis, mania with epilepsy, at the age of 22; died at the age of 24. Menstruation ceased two months before admission. Right ovary, 3 grm.; left ovary, 2·5 grm. No corpora lutea vera seen. Intense congestion. Hæmorrhage into all the follicles. Zona granulosa separated and fragmented. Very marked congestion of discus proligerus, probably the result of status epilepticus. It may be that the congestive stasis of epilepsy would cause follicular destruction.

Assuming that the degeneration of the follicles may arise from two causes: (1) Nutritional, depending upon the quality and quantity of the blood supply to the organ; (2) germinal—the specific vitality of the follicle and especially of the ovum, it is desirable in any future investigation to study particular methods by which the finer histological changes in the ovum can be recognized, so as to determine whether in certain forms of insanity occurring in adolescence, e.g., dementia præcox, a primary degeneration of the ovum occurs, recognizable in the immature follicles.

(*March* 13, 1917.)

Studies on Endocrine Organs of Dementia Præcox.

By M. Kojima, I.J.N. (Tokio).[1]

(Communicated by F. W. Mott, Major R.A.M.C.(T.), M.D., F.R.S.)

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(I) Introduction.

The fact that dementia præcox commences at puberty or in early adolescence may be associated in some way with perverted function of sexual glands. The histological changes in the brain are generally regarded as insufficient evidence to account for the signs, symptoms and progress of the disease. There is reason, from the results of investigations of endocrine organs, for supposing that these organs are associated functionally with the sexual glands and the metabolism of the nervous system. The endocrine organs, especially the sexual glands, by internal secretion may influence, not only the secondary characters, but the mental character.

Many authors have studied the changes of endocrine organs in dementia præcox. Here I give a brief review of recent literature concerning this subject.

In regard to the changes of thyroid in dementia præcox. Amaldi reported a functional insufficiency in two out of four cases. Benigni and Zilocchi found marked sclerosis in one case. Ramadier and Marchand found, in a female case aged 17, marked sclerosis, and the gland weighed 5 grm. In the other four cases they found more or less sclerotic changes, an atrophy of vesicles and great variation in quantity of colloid; while in one case no changes were observed. On the other hand Parhon and others did not find any sclerotic alterations, but

[1] From the Pathological Laboratory, London County Council Asylums.

marked distention of colloid in the vesicles. According to the results of most authors the weight of the thyroid was often lighter than normal. As stated above, the changes of thyroid in dementia præcox are not alike in all cases—viz., in some cases an alteration of hyperfunction, while in others sclerosis connected with hypofunction.

In regard to the changes of parathyroid, Parhon and Urechie found a large number of cyanophil cells in one case, while in the other the gland was in a state of rest.

According to Laignel-Lavastine the pituitary body presents no alterations. Benigni and Zilocchi reported on the contrary some alteration in one out of two cases.

Laignel-Lavastine observed a hypertrophy of the cortex of adrenals, with a tendency to form adenomatous nodules, and an increase of pigment in the zona reticularis. In a case of Benigni and Zilocchi's the cortex was altered.

Parhon and Ghiorghiani have studied the menstruation of 216 female cases, of which thirty-five were dementia præcox. In twenty-five out of thirty-five cases they found amenorrhœa. They consider that ovarian conditions are frequent in dementia præcox, and that they are connected with mental disorders and not with age.

According to Marie and Dide there was no alteration in the testes in dementia præcox. On the contrary Laignel-Lavastine and Vigouroux have found sclerosis with diminution of interstitial gland in several cases. Parhon, Olregia and Urechie found that in the first case the testicle of one side was sclerotic, the interstitial cells were diminished, and the spermatogenesis was nearly absent; while in the testicle of the other side the interstitial cells were numerous, with abundant lipoid, but the spermatogenesis was scarce. They found in the second case equal alteration of sclerosis, an absence of spermatogenesis, but no changes of interstitial cells.

The above is a brief sketch of the results observed by many authors. These changes are multifarious and may not be the effect of mental disorders, because in most cases of the insane complications are found, and these, especially chronic diseases, may cause several changes in the endocrine organs.

During my study in the Pathological Laboratory, Claybury Asylum, under the guidance of Dr. Mott, F.R.S., to whom I desire to express my cordial thanks for his kindness, I was present at a great many post-mortem examinations of the insane, including five cases of dementia præcox, of which two were male and three female. In the case of

a male and two females this condition was combined with chronic tuberculosis; and a male and a female died from broncho-pneumonia after a short illness.

Microscopic examinations of the endocrine organs of these two cases were made, and a description follows.

(II) The Method.

The materials were preserved in 10 per cent. formalin, except the adrenals, which were fixed in Müller-formalin. Some preparations from the pineal glands, thyroids and ovaries were embedded in celloidin, the others were embedded in paraffin. The staining methods used are hæmatoxylin-eosin, Van Gieson's stain, Scharlach or Sudan III, hæmatoxylin and Heidenhain's iron-hæmatoxylin.

(III) Remarks on Case I.

E. S., aged 24, single; former occupation a beer bottler. Admitted to Claybury Asylum on March 28, 1914. First attacked at the age of 23. Clinical diagnosis, dementia præcox. Died on February 19, 1915.

According to the certificates the patient gesticulates and poses. He states that he ought to be in the secret society, but he also states that he does not know what the secret society is. He states that the attendant is his "daddy" and that he came here to see him. The mother stated that the patient had been talking funnily for a year; six weeks ago he gave up his job to better himself. Later he went back and first sacked the foreman and later all the staff. He said he was a millionaire and would make the mother a titled lady.

State on Admission.—Good physique, fair nutrition. Expression is confused; attitude is restless. Respiratory and circulatory organs show nothing special. Teeth carious. Reaction of urine acid; its specific gravity is 1,035. Slight fine tremor of hands. Pupils are medium size, reaction normal. Plantar flexion normal. Knee-jerks are brisk.

Mental Condition.—He often elevates his eyebrows with a restless starting expression. He reacts slowly to questions. There is marked impairment of memory. His conversation is rambling. His method of expressing himself is often slow and there is a tendency to repeat phrases. His remarks are often foolish and incoherent. He is unable to realize his position.

Progress of Case (*March* 30, 1914).—He is weak-minded, and says he wishes to be a doctor. He wishes to join a secret society so that he

can better himself. He is anxious to discover a lady of title who will marry him. He is in good health and condition. He does not improve mentally; is dull and apathetic. He was unable to interest himself in anything.

On February 18, 1915, he was put to bed with temperature 101° F. The next day the temperature was 103° F. His pulse was very poor. He failed gradually and died that day.

Autopsy (made by F. W. Mott, Major R.A.M.C.(T.), M.D., F.R.S.).

A male, fair physique, nutrition good; post-mortem rigidity in hand and arms; lividity on dependent parts of hands and arms; no bruises nor bedsores; no external mark of syphilis.

The skull *nil.* Dura and pia mater nothing noticeable. Pia-arachnoid strips with difficulty. The convolution is complex. No general nor local softening. The pituitary body is small; weighed 0·57 grm. The pineal gland is larger than normal; weighed 0·17 grm. The thyroid is large and pinkish, and weighed 18·3 grm.

The thorax shows normal appearance. The ribs are brittle; the cartilage is completely ossified. The posterior border and apex of right pleura are adherent; there is no fluid. Left pleura is free; no fluid. Bronchi are congested. Right lung is emphysematous. The apex of lower lobe of left lung is solid with red hepatization. Heart muscle firm. Aortic and mitral valves are competent. Coronary arteries patent. Aorta, $\frac{3}{4}$ in. in diameter at the ascending portion. Thymus persistent; weighed 6·5 grm.

The liver is congested and fatty. The gall-bladder contains dark bile: no gall-stones. The kidneys are pale; capsules strip readily. The right adrenal is distinctly yellow; weighed 8·8 grm. The left adrenal shows hæmorrhage in medulla; the cortex is less yellow than normal; weighed 10·7 grm. Intestines and other organs show nothing of note.

The Wassermann reaction of the serum and the spinal fluid is negative.

Cause of Death.—Broncho-pneumonia.

Microscopic Examination.

(1) *The Thyroid Gland.*—The vesicles vary considerably in size and are filled with colloid, which is stained delicate pink by hæmatoxylin-eosin. There is marked increase of intervesicular connective tissue.

The epithelial cells are generally flattened; their cytoplasm contains fine eosinophil granules. In some vesicles a débris of cells is mingled with colloid. There is some thickening of intima of arteries (fig. 1).

(2) *The Parathyroid Glands.*—The three external parathyroids look compact under low power, and a great many watery, clear cells, and a few oxyphil cells are seen. The cytoplasm of the clear cells is not

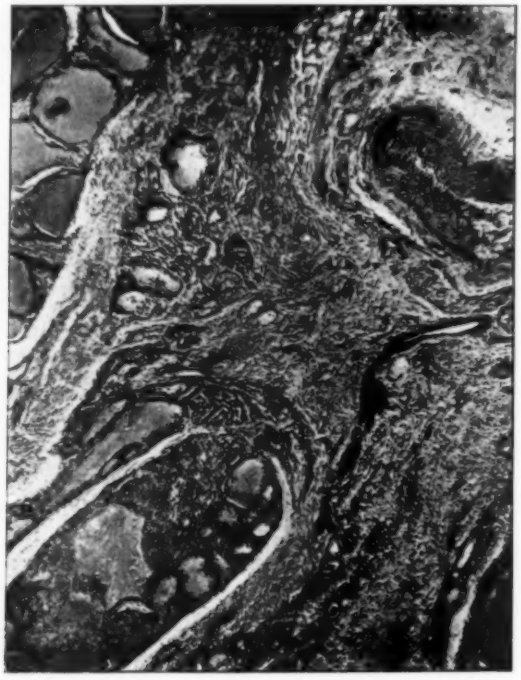

FIG. 1.

The thyroid of Case I, male, showing marked increase of intervesicular connective tissue. The vesicles, which contain colloid, vary very much in size. In some of them a débris of cells is seen. The intima of artery is somewhat thickened. Celloidin. Hæmatoxylin-eosin. (× 60.)

stained by eosin, while the nuclei are stained distinctly by hæmatoxylin. Some of the cells are large. There is no follicular arrangement of the cells.

(3) *The Thymus.*—The remainder of the parenchyma is embedded

in adipose tissue. The cortex is approximately reduced. A few Hassall's corpuscles, which have undergone hyaline degeneration, are seen, enclosed in the epithelial cells of medulla.

(4) *The Pituitary Body.*—The *pars glandularis* is compact. The eosinophil cells are plentiful in the middle. Towards the periphery their number is reduced and the cyanophil and principal cells are on the contrary increased. The connective fibres are increased throughout. A few follicles are seen. The *pars intermedia* is somewhat thickened. In the cleft there is a slight amount of hyaline substance, which is

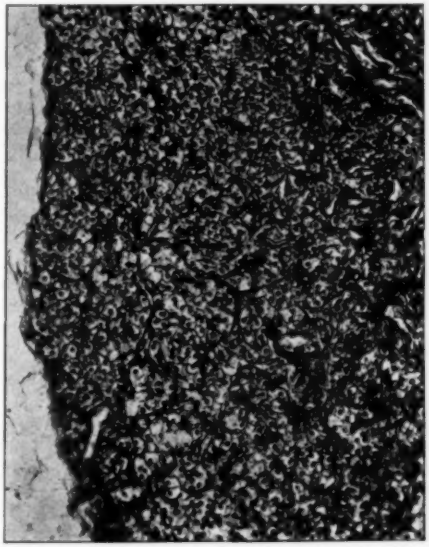

FIG. 2.

The parathyroid of Case I, male, showing watery, clear cells, which occupy nearly the whole section. Paraffin. Hæmatoxylin-eosin. (× 90.)

stained light red by eosin. Nothing noticeable in the *pars nervosa* (fig. 3).

(5) *The Pineal Gland.*—In the hind part of the gland are seen a small cyst and brain sand, which varies in size and shape. On the whole the section shows complete involution.

(6) *The Adrenals.*—The cortex is somewhat reduced in thickness. A few cortex cells contain abundant lipoid substance, which is stained

red by Scharlach or Sudan III, but in the majority this substance is greatly diminished.

(7) *The Testes.* — The connective tissue between the tubules is thickened. The interstitial cells are numerous and contain much lipoid substance, which is stained by Scharlach. Most of Sertoli's cells do not contain lipoid. There is very slight spermatogenesis.

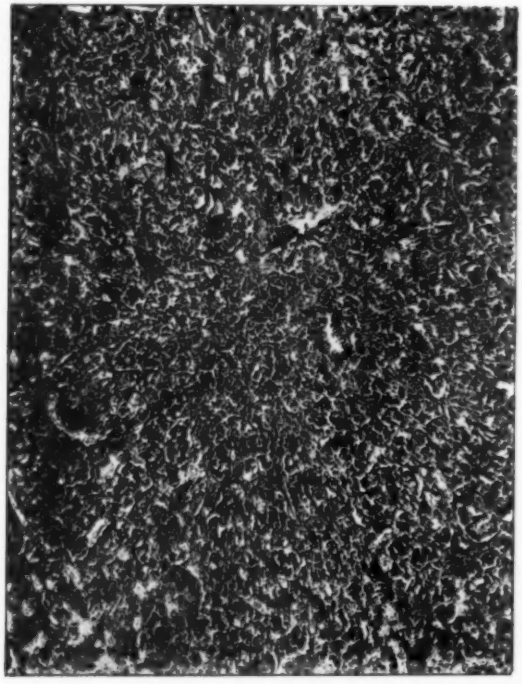

FIG. 3.

The pituitary body of Case I, male, showing an abundance of eosinophil cells. The section looks compact. Paraffin. Hæmatoxylin-eosin. (× 60.)

(IV) REMARKS ON CASE II.

N. D., female, aged 33, single; former occupation, a bookfolder. Admitted to Claybury Asylum on December 7, 1904. First attack when aged 23. Clinical diagnosis, dementia præcox. Died on November 10, 1914.

According to the certificates the patient says that to-day is about November 18, and that she does not know the day of the week. When asked if she is still tired of life she replies that she is sorry for her past sins and wishes to be good.

She went off quite suddenly, became strange and depressed, whereas she was previously bright and cheerful. She tried to commit suicide by throwing herself into the dock. She was taken to the St. George's-in-the-East Infirmary by a police constable and subsequently transferred to the Asylum. A sister at the infirmary says that the patient told her that she was tired of life and had been refusing food. She is very depressed and confused in manner.

Before she had the attack *her periods stopped.* She suffered from anæmia and had taken Blaud's pills for her menses. After she came to the Asylum she complained of pain and recurrence of periods. She recognized her sister, but her sister and her friends noticed that she was quite irresponsible. Occasionally she would smile. Her expression had gone and her hands were cold.

During the first eighteen months her friends had hoped that she would get better, and she looked forward to it. Her sister came one Sunday and instead of finding her in the corridor she was in the ward and a change was noticed. Since then her mind has become worse.

Progress of Case (*December* 12, 1904). — She is suffering from melancholia. She is dull and disinclined to answer questions. States she attempted to drown herself but was saved by a policeman. Admits being tired of her life, and that voices speak to her. She does not know where she is: thinks it is Dartford. Says her maternal grandmother was in Dartford Asylum, where she died; also her paternal aunt is in an asylum. She gazes vacantly about her; is in poor health and condition. November 2, 1911: She is the subject of chronic melancholia. She wanders about in an aimless manner, taking no notice of what occurs around her. There are suicidal tendencies. She rarely speaks. Habits faulty. She is in good health and condition. Her mental condition became worse, and she died on November 10, 1914, after a short illness.

Autopsy (*made by F. W. Mott, Major R.A.M.C.(T.), M.D., F.R.S.*).

A female, well nourished, physique good. Some post-mortem lividity; no external marks of bruises or bedsores; no deformity nor local atrophy; no external marks of syphilis.

The skull is normal. The dura and pia mater have a normal appearance. The pia-arachnoid is also normal. The subdural and subarachnoid spaces show nothing special. The cerebrum is symmetrical. No special marks of brain. The pituitary body is comparatively small, weighed 0·4 grm. The pineal gland is pink, weighed 0·15 grm.

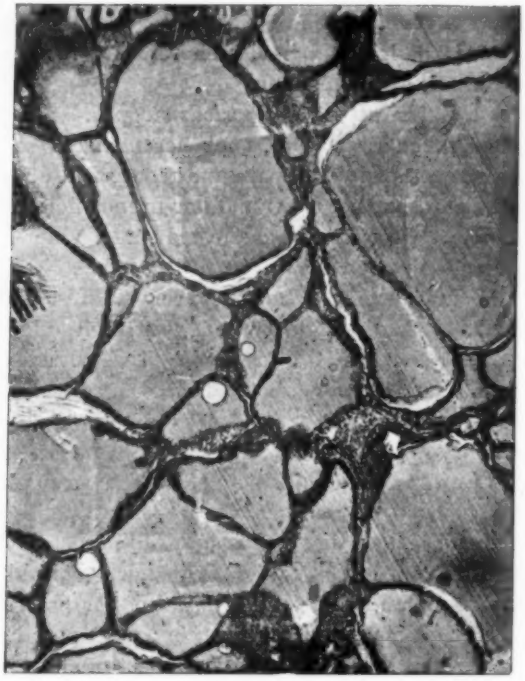

FIG. 4.

The thyroid of Case II, female, showing large vesicles distended with colloid. The epithelial cells are generally flattened. Some vacuoles in the colloid. Celloidin. Hæmatoxylin-eosin. (× 60.)

The right and left pleuræ show no adhesion nor fluid. The bronchi are congested: the bronchial glands show no swelling. The thyroid is small and pink, weighed 7·7 grm.

The right lung shows patchy broncho-pneumonia, especially of the upper lobe, and to a less degree of the lower lobe. Most of the lower

part of the left lung is solid with congestive œdema of the whole lung. All parts float in water. Both lungs have the appearance of having been quite healthy before the onset of the pneumonia. The heart is small; the valves are normal; the muscle has a rather dark œdematous appearance. The aorta shows hypoplasia; it is only $\frac{3}{4}$ in. in diameter just above the sinus of Valsalva.

The liver is congested, normal in size. The spleen is also congested. There is nothing of note in the kidneys and intestines. The adrenals are

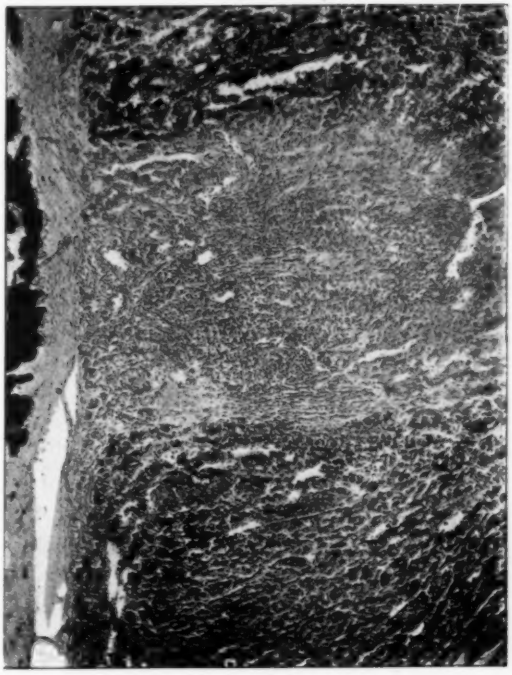

FIG. 5.

The left adrenal of Case II, female, showing a bundle of connective-tissue fibres running through the cortex towards the medulla from the capsule. The lipoid substance, which is shown by dark patches, is scarce. On left extremity adipose tissue is seen as dark spots. Frozen section. Scharlach-hæmatoxylin. (× 60.)

small, the right weighed 2·8 grm., while the left weighed 3·4 grm. The uterus is infantile; ovaries small and atrophied, the right weighed 1·8 grm. and the left 2·7 grm.

Cause of Death.—Broncho-pneumonia.

Microscopic Examination.

(1) *The Thyroid Gland.*—Many of the vesicles are very large, while some of them are small. Generally the vesicles are distended with colloid, which is stained delicate pink by hæmatoxylin-eosin. In the colloid many vacuoles are seen. The epithelial cells are distinctly flattened. Their nuclei are also flat and are stained uniformly dark by hæmatoxylin (fig. 4).

FIG. 6.

The left ovary of Case II, showing an early involution. A few atretic follicles are seen in the photograph. Celloidin. Hæmatoxylin-eosin. (× 60.)

(2) *The Parathyroid Glands.*—The whole section is compact. The eosinophil cells exceed in number the cyanophil cells. There are some follicles, which contain hyaline substance, stained light red by eosin. All the cells have follicular arrangements. The interfollicular connective tissue is somewhat increased.

(3) *The Pituitary Body.*—The *pars glandularis* is generally congested. There are abundant eosinophil cells. The cyanophil and principal cells are far less in number than the former. Follicles, which contain hyaline substance, are seen here and there. A great deal of hyaline substance is seen in the cleft. The *pars intermedia* shows nothing remarkable, but in the *pars nervosa* many droplets of hyaline substance are seen.

(4) *The Pineal Gland.*—The interlobular tissue is markedly thickened. Sand particles, varying in size and shape, are seen here and there.

(5) *The Adrenals.*—There is a striking diminution of lipoid substance in the cortex cells. In many parts of the left adrenal the connective fibres are seen running towards the medulla through the cortex, from the capsule. The medullary part seems to be reduced in thickness (fig. 5).

(6) *The Ovaries.*—In the series of sections of the right ovary there are seen a few corpora candicantia and two corpora albicantia. A few atretic follicles are seen. In the middle a small corpus luteum with a few lutein cells are seen. The left ovary contains a few corpora candicantia and a corpus albicans; a few atretic follicles are seen. On the whole it shows the appearance of undergoing an early involution (fig. 6).

(V) Summary.

The thyroids have an entirely contrary appearance in the male and female—viz., a tendency to hypofunction in the male and to hyperfunction in the female.

The glands are on the whole small, especially in the female. In the male the parathyroids contain watery, clear cells and a few eosinophil cells, and in the female, on the contrary, many eosinophil cells.

The sexual glands and adrenals were very small in the female. The diminution of lipoid substance in the cortex cells of adrenals may be due to the acute disease, as stated by Elliott.

Striking changes are seen in the sexual glands — i.e., very slight spermatogenesis in the testes, and an appearance of undergoing an early involution of ovaries.

In Case II the menses were irregular, as noted in the history.

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PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

J. Y. W. MacALISTER

UNDER THE DIRECTION OF

THE EDITORIAL COMMITTEE

VOLUME THE TENTH

SESSION 1916-17

SECTION OF SURGERY

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1917

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(*October* 31, 1916.)

(Chairman—Colonel C. J. SYMONDS, A.M.S.)

Sterilization of Wounds, Treatment of Suppurating Wounds and Osteomyelitis (Carrel Method).

By WILLIAM O'NEILL SHERMAN, M.D.

AT the outbreak of the War it was quite evident that wound infection was the great problem which confronted the surgeon—practically all wounds, with the exception of those caused by high velocity bullets and small shells, being infected. Carrel began his researches at Tuffier's Laboratory, Beaujon Hospital, Paris; and in collaboration with H. D. Dakin, the Research Laboratory, under the auspices of the Rockefeller Foundation, was established at Compiègne, Oise, in conjunction with Military Hospital No. 21. This work was begun in December, 1914, and completed June, 1915.

The present technique has been used since this date—with certain modifications of Dakin solution and improvements in technique—with remarkable success. Many antiseptics were tried before Dakin solution was finally accepted. It must be understood, however, that Dakin solution is not a panacea or "cure-all," and while it has undoubted bactericidal properties, and is an almost ideal antiseptic solution in that it is non-toxic and non-corrosive, it is not effective unless used with discretion and judgment.

Carrel's first paper, prepared with the collaboration of Dakin, Daufresne, Dehelly, and Dumas, was read by Professor Pozzi before

the Académie de Médecine, on October 15, 1915. He stated that all wounds of war produced by shells, torpedoes, grenades, &c., were infected, and that the methods of treatment in vogue were powerless to prevent gas gangrene, septicæmia, and chronic suppuration. Seventy-five per cent. of deaths after the first twenty-four hours are due to infection (Keogh); 80 per cent. of amputations are the result of infection (Tuffier); 95 per cent. of secondary hæmorrhage is due to infection (Chutro).

The great need was a specific or some technique which would prevent infection, and the variety of methods in use at the present time is sufficient evidence that the treatment of infected wounds is very uncertain, and that previous to the method of Carrel all treatments were generally unsatisfactory. The techniques, as described by Sir Almroth Wright, Colonel Gray, Professor Rutherford Morison, and others, have unquestionably given vastly better results than older methods in use before the War, but the fact remains that relatively few of the patients treated by these special techniques show wounds which have united by first intention—i.e., without pus formation, or healing by granulation. There is a great need for a specific that can be standardized, not only throughout the Casualty Clearing Stations, but also at the Base Hospitals. To accomplish ideal results it is necessary that the public as well as the surgeon should understand that there should be no suppuration, and that if the use of Carrel's method is adopted infection can be aborted or prevented.

To secure the best results the treatment should be applied within the first twenty-four hours. Bacteriological examinations show a variety of microbian flora, anaërobes and aërobes. These are found in practically all wounds, but are usually small in number during the early stages, and localized about the projectile, or clothing, or other foreign material. After the first twenty-four hours the infection spreads rapidly over the surface of the wound, and the microbes are too numerous to count. From this knowledge it follows that it is of the first importance to render wounds surgically sterile in the early stages, and that it becomes more difficult to effect sterilization if the microbes are allowed to spread and penetrate. Therefore all wounds should be freed from foreign material, and the antiseptic agent employed thoroughly during the first twenty-four hours. It is not always possible to remove all foreign material mechanically, so we must rely upon some antiseptic solution which will penetrate into the cavity and surroundings and destroy the bacteria without irritating the tissues or producing toxæmia.

The solution must be in constant contact with the tissues. Dakin solution, technique Daufresne, can be so used that from a clinical standpoint it surgically sterilizes the wound. It should penetrate all the diverticula of the wound, and should be renewed every two hours if complete sterilization is to be obtained. It has the great advantage of being a strong antiseptic, and is only slightly irritating, and can be made by any competent chemist at a very small cost.

Solution.

Original Dakin solution was prepared as follows: 140 grm. of dry sodium carbonate dissolved in 10 litres of tap water, to which 200 grm. of chloride of lime (chlorinated lime) is added, and 40 grm. of boric acid.

Preparation of Dakin solution, technique of Daufresne, as now in use: The solution of sodium hypochlorite for surgical use must be free of caustic alkali; it must only contain 0·45 to 0·50 per cent. of hypochlorite. Under 0·45 per cent. it is not active enough, and above 0·50 per cent. it is irritant. With chloride of lime (bleaching powder) having *25 per cent.* of active chlorine, the quantities of necessary substances to prepare 10 litres of solution are the following:—

Chloride of lime (bleaching powder), (25 per cent. Cl. act.) ...	200 grm.
Sodium carbonate *dry* (soda of Solway)	100 grm.
Sodium bicarbonate	80 grm.

Put into a 12-litre flask the 200 grm. of chloride of lime and 5 litres of ordinary water, shake vigorously for a few minutes and leave in contact for six to twelve hours, one night for example. At the same time dissolve in 5 litres of cold ordinary water the carbonate and bicarbonate of soda. After leaving from six to twelve hours, pour the salt solution into the flask containing the macerated chloride of lime, shake vigorously for a few minutes, and leave to allow the calcium carbonate to be precipitated. In about half an hour siphon off the liquid and filter with a double paper to obtain a good clear liquid, which should always be kept in a dark place.

Titration of Chloride of Lime (*Bleaching Powder*).—Because of the variation of the products now obtained in the market, it is necessary to determine the quantity of active chlorine contained in the chloride of lime which is to be used. This is in order to employ an exact calculated quantity according to its concentration. The test is made in the

following manner: Take from different parts of the jar a small quantity of bleaching powder in order to have a medium sample, weigh out 20 grm. of it, mix as well as possible in a litre of tap water and leave in contact a few hours. Measure 10 c.c. of the clear liquid and add 20 c.c. of a 10 per cent. solution of potassium iodide, 2 c.c. of acetic acid or hydrochloric acid, then put drop by drop into the mixture a decinormal solution of sodium hyposulphite (2·48 per cent.) until decoloration. The number n of cubic centimetres of hyposulphite employed, multiplied by 1,775 will give the weight N of active chlorine contained in 100 grm. of chloride of lime. The test must be made every time a new product is received. When the result obtained differs more or less than 25 per cent. it will be necessary to reduce or enlarge the proportion of the three products contained in the preparation. This can easily be obtained by multiplying each of the three numbers, 200, 100, 80, by the factor $\frac{25}{n}$ in which N represents the weight of the active chlorine per cent. of chloride of lime.

Titration of Dakin Solution.—Measure 10 c.c. of the solution, add 20 c.c. of potassium iodide 1 in 10, 2 c.c. of acetic acid, and drop by drop a decinormal solution of sodium hyposulphite until decoloration. The number of cubic centimetres used multiplied by 0·03725 will give the weight of hypochlorite of soda contained in 100 c.c. of the solution. Never heat the solution, and if, in case of urgency, one is obliged to resort to trituration of chloride of lime in a mortar, only employ water never salt solution.

Test of the Alkalinity of Dakin Solution.—To easily differentiate the solution obtained by this process from the commercial hypochlorites pour into a glass about 20 c.c. of the solution and drop on the surface of liquid a few centigrammes of phenolphthalein *in powder*. The correct solution does not give any coloration, while Labarraque's solution and Eau de Javal will give an intense red colour, which shows in the last two solutions existence of free caustic alkali.

The stock solution should be kept in blue or brown coloured bottles, well corked.

It will readily be seen that there is a difference between the solution of Dakin as originally made and the hypochlorite solution, technique of Daufresne. The original Dakin solution contained 0·5 to 0·6 per cent. sodium hypochlorite—the modified Dakin solution does not contain boric acid, but contains between 0·45 and 0·50 per cent. hypochlorite. Many of the so-called Dakin solutions are not prepared in accordance with the formula of the name they bear, and as a result the treatment

has been condemned in cases where some other solution has been used in the name of Dakin.

The method of application—technique Carrel—must be followed if successful results are to be secured. It is possible that Dakin solution represents but 20 per cent. of the cure, and that the technique of Carrel represents 80 per cent., that the success of this treatment is dependent upon the thoroughness with which it is applied and the care given to even the smallest details of the technique. The surgeon-in-chief, as well as his assistants and the nursing sisters, should have a mutual understanding with reference to this treatment, and work in hearty co-operation in order to bring about technical perfection. It must be repeated that much of the success of the treatment is dependent upon the intelligence with which it is applied by the nursing organizations, and they should be given special instruction. When handling wounds of war it is not always possible to give the treatment that might be desired as many obstacles are encountered which prevent the carrying out of necessary details.

Method of Application.

(1) *Dressing at the Advanced Dressing Stations.*—The field should be disinfected with tincture of iodine. If the wound is small or narrow an injection of Dakin solution into the course of the wound should be made ; if wide and freely open, a gauze pack saturated with Dakin solution should be applied. While this dressing is not absolutely necessary it materially aids in the prognosis.

(2) *Dressing at the Casualty Clearing Station.*—If the wounds are extensive the patient should be given an anæsthetic and the operating field prepared in the usual way. All wounds should be explored with free incisions and all foreign material removed, but it is not necessary to scrub or swab the wounds. All bleeding points should be ligated. The wounds should be thoroughly examined for foreign bodies.

Before the dressing is completed the solution should be injected into the wound to ascertain if it is reaching all parts and to determine the quantity necessary to completely fill the cavity. For the final dressing, sheets of gauze surrounded with non-absorbent cotton have been found to be the most useful. Turkish towelling liberally used is also satisfactory as a final covering.

Apparatus.

Rubber tubes, 6 mm. in diameter and approximately 25 cm. long, are used, tied at one end, and beginning from the distal end a series of small holes are pierced at intervals of ½ cm. for perforation—six, eight, or ten perforations usually being sufficient. The number of tubes to be used must be decided by the size and nature of the wound. They should be introduced into the cavity so as to allow the solution to be well sprayed. The tubes should be kept in position by means of strips of gauze loosely placed between them. Gauze should not be packed into the wound. A gauze pad, saturated with Dakin solution, is used to cover the wound. A graduated container is preferable, and should be elevated not more than 3 ft. above the head of the patient, but where pain is experienced by the patient the container should be lowered.

The non-absorbent gauze dressing should be approximately 3 cm. thick and of various sizes, and go once around the limb. A tube covered with Turkish towelling may be used for superficial wounds. Such a tube has been found to remain in position and keep the areas sufficiently bathed without slipping about over the tissues. A strip of gauze should be placed between the tubes before covering with Turkish towelling, to keep them in position. The antiseptic solution should be introduced into the wound every two hours by the nurse, who releases a stop-cock or controlling valve, allowing just sufficient solution to enter the wound, bearing in mind that it should not be irrigated, but simply filled.

The most effective method is the intermittent instillation. The nurse soon learns by practice just how much solution should be injected into each wound, and again it should be remembered that it is important that a fresh injection should be given every two hours.

Re-dressing. — Every antiseptic precaution should be exercised, as wounds can be re-infected very easily, even after sterilization. Vaseline gauze should be used to protect the skin, and after this is removed the skin should be cleansed with ether and swabbed with a solution of sodium oleate. This solution is neutral and non-irritating. Wounds are sponged with this solution to remove the wound secretion, sloughs, or other free débris. The Carrel tubes are again introduced as in the original dressing. The skin should be protected against the possibility of burning by the application of gauze saturated with vaseline. Re-dressing should be done every day, if possible, but in cases of emergency it can be delayed for two or three days if the wound is kept bathed

with solution. It is essential that a free and open incision should be made to permit the easy introduction of the tubes and the gauze.

Suture and Cicatrization.—When the clinical appearance of the wound, together with the bacteriological verification, indicate that it may be safely sutured by interrupted silkworm gut, the edges should be excised and if granulation tissue is present it should be removed. If the tension is too great lateral incisions should be made to allow the edges of the wound to be brought together. Where there has been great loss of tissue it will be found impossible to suture. Such wounds can be closed by adhesive plaster strips, followed at a later date by plastic operations if necessary. Ninety-nine per cent. (Carrel) of wounds which have been thoroughly sterilized have united by first intention with practically no reaction either along the line of incision or at the stitches. There is no drainage from the wounds—they very much resemble post-mortem wounds.

Treatment of Acute and Chronic Osteomyelitis and Epiphysitis.

Free incision should be made, and the necrotic tissues gently excised. The Carrel tubes should then be introduced in the manner previously described and dressing applied. Within twenty-four hours the dressing will become saturated with a discharge, giving it an inky appearance, and making re-dressing necessary. This discharge rapidly decreases and disappears within a few days, the wound taking on a healthy appearance. The introduction of Carrel tubes in a sinus leading to necrotic bone will not sterilize either bone or soft tissues, but such a wound can generally be sterilized within fifteen to twenty-five days and closed with adhesive plaster.

Compound Fractures with Deformity.

After wound and bone have become aseptic a plate can be used, if indicated, to hold the fractured part in position. If it has been found necessary for the wound to be kept open with the Carrel method the plates and screws will probably require removal after consolidation has taken place. There is not sufficient available clinical evidence at the present time to state whether or not these wounds can be closed, either at the time of the application of the bone plate, or at a later period when the wound has become aseptic. Where plates have been used and wound infection takes place, the wound should be freely opened

and Carrel tubes inserted. Vandervelde (service of Depage) intends either to plate or wire fractures immediately where the operation is indicated, and then follow with the Carrel method. If the wounds are rendered aseptic in the early stages it will permit of early operations, such as bone-grafting, plating, nerve repairs, and plastic operations, without the danger of re-infection.

Sterilization of the Uterus, Gall-bladder, Urinary Bladder, Localized Abdominal Abscess.

Uterus.—The infected secundines are carefully removed. The cervix is well dilated and the hard rubber dilator and pessary is inserted into the cervical canal. The dilator extends from the external os to the internal os. There are five perforations in the dilator to permit of the entrance and exit of two rubber tubes which are passed into the uterus. They are connected with a four-tube glass distributor, which is attached to the irrigator. The irrigator is placed 3 ft. above the head of the patient. Rubber sheeting is attached to the external surface of the dilator at the external os, completely enveloping the rubber tubes and central gauze drain which emerges through the central opening of the dilator. Ribbon gauze is lightly packed into the uterus. This gauze serves the double purpose of holding the tubes in position and retaining the Dakin solution. The tubes should be changed every second or third day and the gauze removed daily. The vagina and cul-de-sac should be gently packed with just sufficient gauze to keep the dilator in position. Dakin solution is injected into the wound every hour, if necessary, the amount to be injected depending upon the pathological condition existing. A large absorbent pad is applied to the vulva to absorb drainage.

Modifications of this technique can be used for the gall-bladder and urinary bladder, pleural cavity and abdominal abscesses. In the gall-bladder, pleural cavity, and in the urinary bladder, vacuum drainage will probably be of assistance together with intermittent injections of Dakin solution.

Bacteriological Verification Relative to the Suture of Wounds.

Depage states that the transformation which occurred in their cases was very impressive, that immediate complications became more and more rare, and that suppurations disappeared completely, so to

speak. Where it is possible, bacteriological verification is a most valuable feature of Carrel's method. This verification consists in determining the number of microbes present in the wound during the stages of its evolution. A smear is prepared according to the usual laboratory methods and the number of microbes contained in the microscopic field is counted. These smears can be made by a doctor, or by a nurse who has received special instruction.

During the first six or seven days smears show that the exudation consists of enormous numbers of microbes and polynuclear leucocytes. After the seventh day the polynuclears gradually disappear, and in their place small mononuclears appear, the microbes decreasing at the same time. After the tenth or eleventh day macrophages appear, and but few bacteria are now found in the field. The bacteria are incorporated by the phagocytes. The appearance of macrophages may be construed as a favourable symptom and indicates that the wound is becoming aseptic.

In order to follow the progress of infection a bacterial chart—reminding one of a temperature chart—has been devised. In a general way the bacterial curve, treated by the Carrel method, follows a more or less characteristic course. In the virulent infections as many as 1,000 microbes are counted in a single field. The chart may indicate more or less marked oscillation, but nevertheless the general tendency is towards descent of the bacterial curve, which arrives at zero after five to twenty-five days of treatment. Superficial wounds are much more rapidly sterilized, and can be sutured at the end of from six to ten days, but extensively soiled and infected wounds require a longer period to become aseptic. In cases of fracture extreme care should be taken to obtain smears from different parts of the wound before suture is attempted. Compound fractures are generally sterilized within fifteen to twenty-five days. Loose bone or necrotic tissue should be removed.

It must be remembered that a wound rendered aseptic by the Carrel method can be rapidly re-infected by errors of technique or if the treatment is suspended. While wounds treated by the method of Carrel are surgically aseptic, they are not bacteriologically aseptic. Smears should be made every two or three days, and if the bacterial count has been reduced to an average of one bacterium to five fields, counting at least twenty fields, and this result obtained for five successive days, suture may be accomplished empirically. It has been found that if wounds are sutured while the ratio is one bacterium to one field or to two fields there is a tendency towards stitch abscess.

Cocci are the most important bacteria and are the first to be removed from the field. Methylene blue or *thionine phenique* are very satisfactory stains.

Results.

Depage reports 137 sutures as follows:—

(*a*) One hundred and twelve complete successes. In these cases reunion was perfect in the whole extent of the section without there being the least inflammatory reaction.

(*b*) Twenty-three partial results. In this category are placed two cases in which the stitches came loose, either in consequence of necrosis of the section or of too strong tension, or on account of slight suppuration which resulted from an insufficiently established asepsis. This result, instead of detracting from the value of bacteriological verification only confirms its utility.

(*c*) Two failures. One in the leg with exaggerated tension of the skin. This case should not have been sutured owing to tension, and it was afterwards treated by strips of adhesive plaster and rapidly recovered. The other failure was a fracture of the radius with a large wound and tension of the skin. It was necessary to reopen the wound the next day on account of recurrence of infection in the soft tissues. The wound was rapidly sterilized afterwards, and the open fistula completely closed within a few days. The fracture was cured as if it had been a simple one.

The nature of these wounds was as follows:—

(*a*) One hundred and two wounds of the soft tissues.

(*b*) Six articular wounds, consequent upon resection of the knee and the left fibula left widely open: five complete successes and one partial result.

(*c*) Twelve wounds of amputation stumps: ten complete successes, two partial results.

(*d*) Seventeen fractured wounds: fifteen complete successes, one partial result, one failure.

In conclusion Depage states that since July 1, 1916, they have had under treatment in their hospital 103 cases of fracture. At the time of my visit to him at La Panne (August 20, 1916), there were eighty cases of compound fracture undergoing treatment; not one was suppurating, and all were as healthy as simple fractures.

Dr. Dehelly and Dr. Dumas report the treatment of 155 wounds from shell splinters, grenade splinters, bomb and mine splinters.

Wounds caused by bullets are not included in these statistics, and of these 155 wounds, 135 were closed—i.e., 87·4 per cent. Out of the 135 closed up wounds, 121 closed before the twelfth day—i.e., 89·6 per cent.; 119, or 88·25 per cent. were healed up in less than thirty days, and of these 155 wounds twenty-five were complicated by fractures. Of these twenty-five cases twenty-two were closed and eighteen were healed in less than thirty days. Similar results have been secured by Pozzi, Tuffier, and Uffoltz.

Carrel has made a number of exhaustive experiments with chloramine paste, in which sodium stearate is used as a base. This paste should only be used for superficial wounds. Considerable difficulty was experienced in securing a paste which would give up the chloramine to the tissues. The general appearance of wounds paste-treated is totally different from those treated by the solution of Dakin, but bacteriological examinations show that the results obtained are identical. The paste is used to simplify the method of dressing.

Wounds have frequently been found to be re-infected from skin infections, so that care must be exercised to prevent contamination of the wound from this source. Every precaution should also be taken to prevent infection occurring at time of operations and dressings.

Contrary Indications.—It should never be forgotten that the solution must not be heated. It should never be applied to or used in the eye, in the general abdominal cavity, or intravenously, because of its hæmolytic action; several deaths have resulted from intravenous injections. It should be kept in a cool place, free from exposure to light. It should never come into contact with alcohol.

Humanitarian and Economic Features.

It has been demonstrated beyond all question of doubt that the great majority of wounds can be closed by suture and without suppuration: thereby many of the sequelæ are avoided, and the period of convalescence of the wounded is materially reduced. Under the treatment which I have had the honour of describing, the stay of the wounded in hospital is very appreciably shortened, and many now leave in from four to six weeks who would have required not less than from three to six months' treatment under former methods. It minimizes the effects or even prevents the onset of atrophy, ankyloses, adhesions, and septicæmia. It obviates the necessity for amputation, and lessens the number of deaths. Professor Chutro (service of Gosse) states he is now

doing one amputation for sepsis and hæmorrhage where formerly twenty were necessary, and where there were ten deaths there is now only one. These statements are startling, but they bear investigation and demand that every consideration should be given to them from both humanitarian and economic motives. The loss by death, or from permanent or partial disability, increases the economic value of every remaining individual. Decrease in earning power is in proportion to the permanent disability sustained, and the machinist or skilled mechanic who suffers the loss of an arm becomes doubly a loss to his community—first by reason of the pension to which he is entitled, and secondly owing to the diminution of his productive capacity.

The employment of Carrel method and Dakin solution shortens convalescence and minimizes pain; it appreciably reduces the cost of hospital maintenance and the strain imposed upon doctors and nurses. Further than this, it is at once a privilege and a duty to employ all means of alleviating the pain and disabilities of the wounded.

I cannot conclude this paper without attempting to express the very deep gratitude I feel towards Dr. Carrel, Professor Depage, Professor Pozzi, Professor Tuffier, and Professor Chutro, for the unfailing kindness which they extended towards me. This kindness enabled me to obtain a knowledge—hitherto undreamt of—of their wound treatment. The facts placed before you speak more eloquently than any words in my power of what is being accomplished by their efforts, and I esteem myself happy to have the honour of helping to disseminate this knowledge. Also I wish to take this opportunity of expressing my sincerest thanks to Sir Arbuthnot Lane, Sir Berkeley Moynihan, Sir Alfred Keogh, and Sir William Osler, as well as to Mr. E. M. Corner, for the kindness they have shown me since my arrival in England.

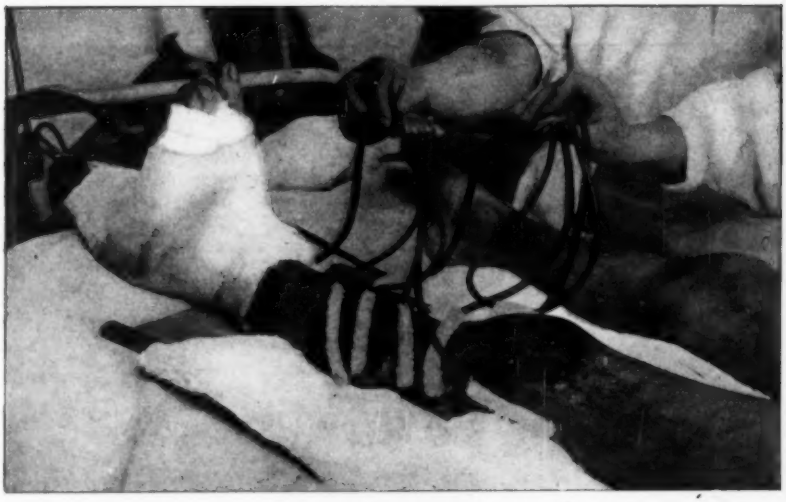

FIG. 1.

Method of application of Turkish towelling tubes to wound. The glass distributor with multiple nozzles is seen, together with the main tube from the irrigator reservoir.

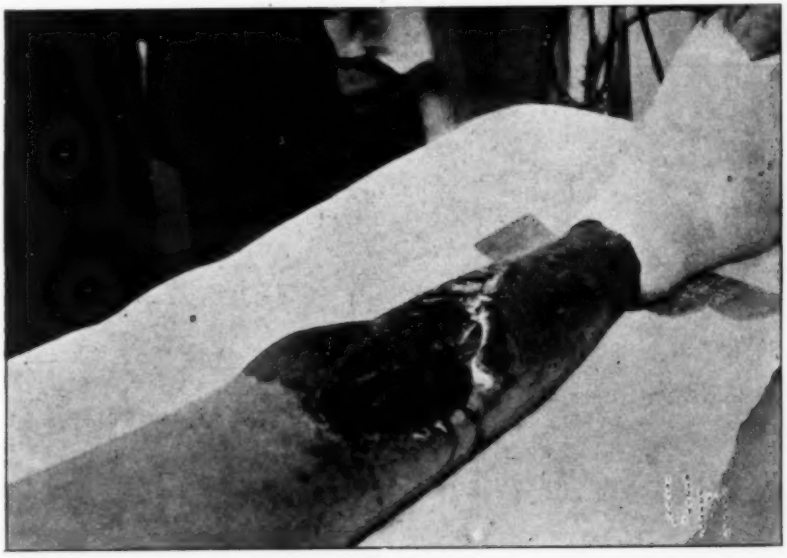

FIG. 2.

This shows the same case as shown in fig. 1, a compound comminuted fracture of tibia and fibula, with extensive laceration of the soft parts. This wound was rendered sterile in twelve days.

FIG. 3.

This shows the use of tubes where a counter incision is made.

FIG 4.

Case of infection following resection of the knee-joint which was sterile in seven days after beginning the treatment.

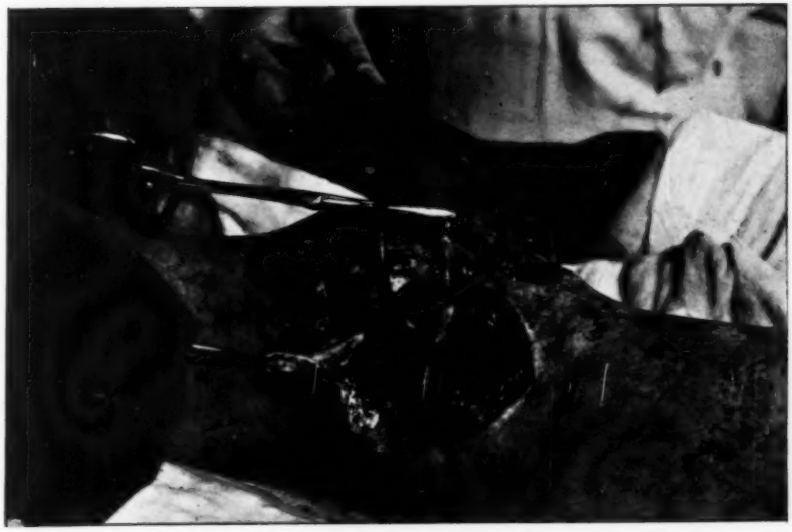

FIG. 5.

Wound of the knee-joint with application of Carrel tubes six days after the injury.

FIG. 6.

Resection of head, neck, and upper two-thirds of humerus. Admitted six days after gunshot injury and clean after eight days of treatment.

FIG. 7.

Injury of jaw and soft parts of the face.

FIG. 8.

Same case as preceding, after short period of treatment.

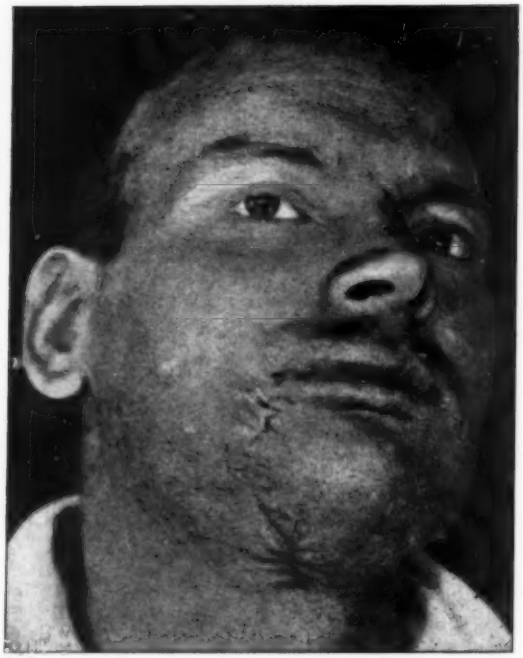

FIG 9.

Final result of case shown in figs. 7 and 8 after application of Carrel treatment.

FIG. 10.

Compound comminuted fracture of head of humerus, with extensive laceration of soft parts.

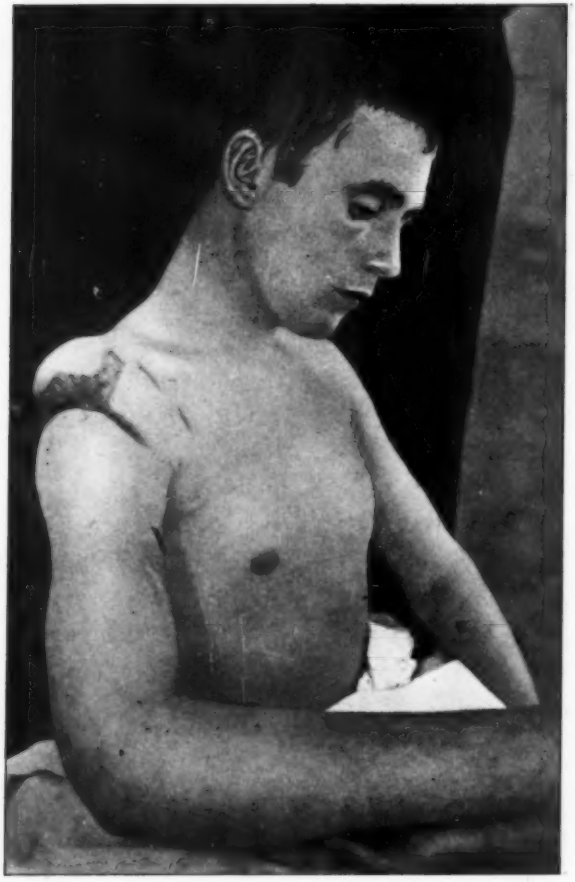

FIG. 11.

Same case as preceding, later in the treatment.

FIG. 12.

This shows final result of the case shown in figs. 10 and 11 after 2½ months' treatment by Carrel's method.

FIG. 13.

Gunshot fracture of femur; wounded July 1, 1916; sterile on July 15; closed July 17; consolidated August 14, 1916.

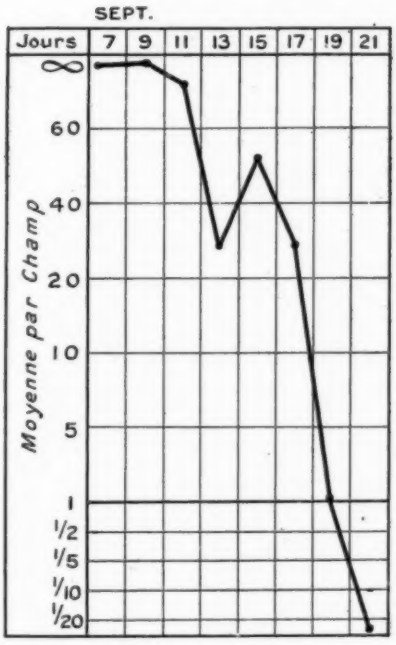

FIG 14.

Microbial chart showing bacterial curve of a suppurating wound. The symbol ∞ stands for infinity, and below this a decreasing number of bacteria to a field. Below the line the figures show the number of fields viewed to find one bacterium.

DISCUSSION.

Surgeon-General Sir WATSON CHEYNE, Bt.: If no one else is prepared to say anything, I would like to say on behalf of myself and the meeting generally, that we are extremely indebted to Dr. Sherman for his very interesting demonstration. I had an opportunity of seeing a few of his specimens before, but I have been extremely interested in seeing more. I think we may say that he has shown us—I do not say in every case, but in a very considerable number of cases—results which have not been obtained before. The most important one is that a compound fracture, received in a highly septic condition, can be so completely sterilized by Dakin and Carrel's treatment that at the end of fifteen to thirty days—I have not particularly studied the number of days, but at the end of a certain period of time—it can be stitched up and the wound heals by first intention. That is a very remarkable result, and one which the ordinary method of treatment will not give. And it is a fact which pleases me very much, because as you know, I have tried to demonstrate that the only way in which you are likely to get a good and rapid result in these cases of bad gunshot wounds, is to employ antiseptics, not anyhow, but with the single view of destroying the bacteria which are in the wound at as early a period as possible. I think Dr. Sherman's demonstration shows that that can be done even in cases not received till sepsis has been established, which is a most important point. I have myself had little practical experience of Dakin solution. As a matter of fact I went out to the Dardanelles last year with Dr. Dakin with the view of studying this solution and its effects, but I was taken seriously ill and had to come home at once, and as I have only quite recently been able to resume work I have done very little with it. I used it, however, in two cases at the Dardanelles, both of them pretty bad. One was a gunshot wound penetrating the knee-joint, and one was a flesh wound of the thigh. Both remained aseptic from the first, both bacteriologically and clinically. One of the great difficulties which stand in the way of the introduction of such a treatment universally—the greatest, to my mind—is the difficulty of keeping a patient under the same doctor all the time. Carrel has the advantage which I suppose very few others have had, that he has been able to keep a patient under his own treatment for any number of days that he chooses. If on the other hand hospitals are being constantly evacuated and the patients transferred from one surgeon to another there is no continuity in the work and no method of treatment has a chance of being thoroughly tested. This is too long a matter to go into, but some means would need to be devised by means of which the patient either is retained near the Front if badly injured or a series of teams are established of men of the same way of thinking so that the continuity of treatment is maintained.

Captain JOCELYN SWAN, R.A.M.C.: I also think we are very much indebted to Dr. Sherman for his demonstration. Working as I have been practically since the beginning of the War in an English base hospital, I can

only speak of the experiences that we have in the treatment of these wounds purely from the standpoint of the work of an English base, not from that of the immediate treatment which can be given to cases in France. Still, we have a large number of cases which reach us within a few days of the receipt of the wound, and I must honestly say I feel that a very great deal of the sterilization of these wounds is obtained as much by the mechanical effect of the fluid used as by the virtues of the fluid itself. Personally, I think one is able to get the best results by a combination of the hypochlorites with hypertonic salt solution. I am myself in favour of using hypochlorites very extensively, and by the flushing method which Dr. Sherman has described; but I think I have seen better results when cases have been treated intermittently, partly by hypochlorites, partly by hypertonic salt solution. With regard to the cases arriving in England which have been sewn up in France, I am afraid my experience of them has been somewhat unsatisfactory. We do get a certain number of the cases which have been treated by excision of the wound and suture, in which we have to open the wounds rather freely and drain them, because of the suppuration which has taken place inside, but I have no means of telling what method has been adopted to sterilize the wound. At the same time, I have treated a large number of wounds by obtaining, I will not say exact sterilization, but a state in which there were healthy granulations over the wound, and have then sewn them up by undermining the tissues and secondary suture. I have been very pleased with the results I have obtained in this way, having treated them in the first place by hypochlorites and hypertonic salt solution. I think there is yet much to be learned in regard to the treatment of these septic wounds and very much concerning the method of application of each treatment. The question of the treatment of superficial wounds is a most important one. One point which Dr. Sherman mentioned was that compound fractures could be plated after sterilization of the wounds by this method. That, I consider, is one of the most important points in his paper. I am convinced that if these wounds are freely opened, drained and treated with hypochlorite solution and saline until the wound assumes healthy granulations, then plating of the fracture is a safe proceeding. I have carried it out in a large number of cases, and with very good results, not only with regard to good alignment and functional use afterwards, but also without the necrosis and the sequestra which one so frequently sees in fractures due to gunshot injuries. It is a point I would lay stress upon, that after such treatment of gunshot fractures complicated by septic wounds, they can be plated with success. I have made no attempt to suture these wounds after plating but leave them freely open to granulate. There is entire relief from pain, dressing the wound is simple and the plate is removed after about five weeks. From Dr. Sherman's remarks it would appear that not only the superficial but the deep aspect of a wound can be rendered sterile by the intermittent flushing with Dakin solution. I think that sterility, in the sense we use it, can be rarely obtained, for in a very large number of cases in which bacteriological investigations have been carried out

in my cases, the deep recesses of the wound almost always contain organisms—frequently anaërobic gas-forming organisms. However, if as Dr. Sherman maintains, these wounds can be closed with such a large percentage of success, it will materially shorten the period of convalescence of the wounded and be an immense saving in the expenditure of dressings, both of which are highly to be desired.

Colonel C. J. SYMONDS (Chairman): As no one else rises, I would like to say a word or two. I have already expressed my own indebtedness to the gentleman who read this paper. We must not go away with the idea that everything is new in the methods or technique of which he has told us to-day. For instance, the method of using irrigation tubes in wounds has now been established some time, also the method of tying one end of the tube leaving the lateral holes for the distribution of the fluid is used a good deal. We were using it largely at Malta, and it is in extensive use at Netley. Therefore, as far as that goes, I am not inclined to admit that there is anything new in the technique. With regard to the use of antiseptics instead of saline, I agree with Sir Watson Cheyne and Dr. Sherman that we shall get better results from adopting this plan than we do with using pure saline. With regard to the excellent results which he has shown us, I take it that if you look at all those pictures and photographs, it comes to this: that where the wounds are on the surface, like for example, those he showed us in which the wounds were on the shoulder, on the buttock and the thigh, sterilization is easy, and it does not very much matter what you use. I should very much hesitate to attribute the whole of the credit to the hypochlorite solution in that type of case. Nor do I think that the hypochlorite is the only method which will produce a nice healthy wound such as you can safely close in three weeks, because other things will also do it; permanganate of potash will do it, perchloride of mercury will do it, saline and eusol (?) will do it. With regard to that type of wound, therefore, I cannot accept anything of this as quite new, and I cannot accept it as a better result, though it may be quicker, than some of our present methods. In these cases we have closed with strapping, and surgeons are getting very good results. The wounds are sutured up when they become clean and show a tendency to heal. Still our results are not so speedy as those which Dr. Sherman has just described. When we come to the consideration of compound fractures, however, I must say I am prepared to give full credit to the great improvement which he has demonstrated, because those are cases which do trouble us very seriously. But of course there is this great difference; as a French surgeon said, "There is a surgery of the Front and a surgery of the Base," and we in England are very much at the Base; we get our cases when they are very profoundly infected, and we have special difficulties on that ground, and we cannot close our wounds quite so early. But after what I have heard to-night I shall certainly endeavour to inculcate this method at Netley, where I have been attached since the end of July. Irrigation methods have been established and carried

out, and I may say our wounds are doing extremely well on the whole, but not so well as those which Dr. Sherman has described. I expect you will agree that whatever method you are going to carry out, a very great deal depends on the belief you have in the method. It is very difficult to inculcate a belief like that. Many of these results, like those obtained in fractures, will depend on the man who does it. Hence we have our special difficulties because we are constantly changing our medical officers. When you can have continuous attention by men thoroughly believing in the method employed, you will, of course, get better results than in other conditions. Those are some of the difficulties that we have to deal with. However, I am very glad I was able to come up to-day and take the chair on this occasion, because I may have the opportunity of introducing this method among the many hundreds of serious cases. I am grateful to Dr. Sherman for what he has told us. I want him to think that my criticisms are natural and healthy, because the methods of application of the remedies are not altogether new. And here we come to a very great difficulty: Can we get this material? I am told it is extremely difficult to prepare it, so as to ensure that we get 0·45 as a minimum, up to 0·50 as a maximum of alkalinity. I hope he will be able to tell us that this material can easily be obtained. With regard to joints and some of the deeper wounds, the results we are getting by the employment of rest and irrigation are very good indeed. I gather from Dr. Sherman that Carrel's method is intermittent irrigation, as opposed to the continuous drip. I am satisfied having tried it for one and a half years, that the continuous drip is not so good as intermittent flushing. I think that in knee cases and deep wounds we are getting better results with that than with the continuous drip. However, anything we can get which will make these wounds heal we all accept, I am sure, and are prepared to try; and one is only jealous of the splendid results which have been obtained. I think the result of this contribution will be to fire us with enthusiasm to try and equal them if we can. If, Dr. Sherman, you think you have anything to reply to, beyond the meeting's cordial thanks, which we hope you will convey to Dr. Carrel for us, we shall be glad to hear you.

Dr. SHERMAN (in reply): I think it would be most unfortunate if we assume the attitude that there is nothing new in this solution, or in the method of using it. Since July 17 I have visited seventy-five hospitals. I have seen six surgeons who were using a modified Dakin solution and who have followed out the technique of Carrel. I have seen ten or fifteen others who, in effect, have said "Yes, we are using Dakin solution and using the Carrel method, and we don't think much of it." May I give a personal illustration? I visited a very eminent surgeon before leaving France. I said, "I have come to tell you whom I have seen in Compiègne and La Panne: three or four very capable surgeons who tell me they cannot understand why you are opposed to the Carrel method." He replied, "Let us sit down and talk it over," and we did. At this particular hospital there were,

I think, 150 compound fractures, and they were all infected, they all had pus in them, and they were running temperatures, entirely contrary to what was the case at Compiègne and La Panne. We discussed the cases and examined the solution. I said to the chemist "What strength hypochlorite are you making or putting into this so-called Dakin solution?" He said, "I don't know." "How did you make it?" He went through the old process, the one which was employed one and a half years ago. There is a difference in the preparation of the original Dakin solution and that which is now in use in France—a marked difference. This eminent surgeon had a number of bacteriological experiments made with this so-called Dakin solution, and the bacteriologist stated that all bacteria thrive in a 50 per cent. solution, that it had no more bactericidal virtue than Thames river water. Of course it did not, he did not have hypochlorite solution. We are making a great mistake if we are going to say there is no difference between these different forms of treatment, because there is. Let us analyse the work of Depage, Chutro, and Tuffier, men who have treated not one but thousands of cases: they have gone all through the different techniques, they have tried them all, and there is no possible comparison between the results. It is impossible to distinguish the difference between an infected and a non-infected wound by the naked eye. It is the bacteriological chart which tells the story. A wound which looks infected may be sterile, while a wound which seems to be clean may be infected. Certainly both Depage and Carrel have received a great many of their cases within twenty-four hours of the infliction of the wound; but as I have shown one case came to Carrel nine days after he was wounded, in a condition which seemed to indicate amputation of the forearm, but it was sutured on the eighteenth day and saved. Chutro receives his cases—i.e., infected—five to seven days after wound is received. It is going to be most unfortunate if we say there is nothing new in the solution or method. Carrel has undoubtedly proved his case. The crux of the situation is this: Can we suture and have primary reunion in 99 per cent. of our cases when the wounds have been sterilized with the other methods? It is possible with the Carrel method. Certainly the results of Depage, Chutro, Tuffier, and others are worthy of thoughtful consideration.

Section of Surgery.

President—Surgeon-General Sir ANTHONY A. BOWLBY, K.C.M.G.

Chairman—Colonel C. J. SYMONDS, A.M.S.

(*December* 13, 1916.)

A Clinical Study of Anaërobic Wound Infection, with an Analysis of 107 Cases of Gas Gangrene.

By FRANCES IVENS, M.B., M.S.[1]

IN this paper I do not propose to deal with all the septic complications of war wounds, but more especially with those associated with the presence of gas-forming anaërobic organisms. My notes will be founded on a personal observation of the cases admitted to the Scottish Women's Hospital, Royaumont (Hôpital auxiliaire 301), from January 13, 1915, to October 30, 1916. During this period 2,267 French soldiers were admitted, of whom 1,694 were wounded, 344 were surgical cases, and 229 were sick. Out of 1,694 wounded, 464 showed signs of anaërobic wound infection, and 107 suffered from gas gangrene.

The hospital, which consists of 400 beds, is near to a military evacuating station in the Army zone, through which a large number of wounded pass, and where it is necessary to take out of the ambulance trains many urgent cases unable to travel further. It is possible, therefore, that an unusual number of cases of gas infection have come under treatment, and that these numbers in no way indicate the general incidence.

[1] Surgeon-in-charge, Hôpital auxiliaire 301 (France).

It has been noted that after every attack cases of gas gangrene occur, and the highest level was reached on July 2, 1916, when, out of 127 wounded admitted during the twenty-four hours, gas infection was present almost without exception. Many cases needed immediate amputation, and some were already septicæmic. On this account and because of the urgency of the cases, it has been specially difficult to obtain the complete and accurate note, the successful X-ray plate, and the fully worked-out bacteriological report, so desirable for a complete study of this important subject. It is usually necessary to operate upon the patient as soon as he can be prepared, and an X-ray examination made. It is impossible to wait for the result of cultures from the discharge and blood. Our bacteriologists were good enough, however, to examine immediately smears from the wound and muscle discharge, and to give a provisional diagnosis based on the presence in the films of many Gram-positive bacilli. It was remarkable in how many instances this bacteriological black list coincided with the cases sent up for immediate operation from the wards. This is not surprising, as we know that all pathogenic agents of gas gangrene such as *Bacillus aërogenes capsulátus* (Welch), *Bacillus perfringens*, *Vibrion septique*, *Bacillus œdematiens* (Weinberg and Séguin), and *Bacillus fallax* (Weinberg and Séguin), are all Gram-positive when examined in the discharge fluid. It is also possible that after further observations some help in coming to a decision about treatment may be derived from a minute study of the X-ray plate, as has been recently demonstrated by Dr. Agnes Savill,[1] which shows us not only the projectile and fracture, but a special tracery, streaking or striation, associated with the presence of gas in or between muscle fibres. Some special characters even help us to differentiate between the fatal *Vibrion septique* and the more innocent *Bacillus sporogenes*. The fact that these gas organisms are rarely present alone makes any differentation extremely complicated, but the appearances are interesting and suggestive when associated with complete bacteriological study.

It is in the border-line cases that we need most help, for if the infection is deep-seated and clinical signs are not obvious, one may be tempted to defer amputation until too late. The X-ray plate may show us that a virulent infection is present in the deep muscles, which demands radical treatment while still localized. Quite recently in a calf wound, which to all appearances was superficial, the X-ray plate

[1] A. Savill, "X-ray Appearances in Gas Gangrene," *Proc. Roy. Soc. Med.*, 1916-17 (Sect. Electr.), p. 4.

showed gas tracery of the deeper muscles, and after cutting away the gangrenous superficial tissues I explored through the deep fascia, where I found an unsuspected but advanced stage of muscle infection.

I regret that circumstances have not permitted us to make regular examinations of the blood by means of blood counts and films, as I believe that the presence or absence of leucocytosis might be an aid in prognosis and treatment. In the only case of gas septicæmia which I had an opportunity of examining before the War, the blood showed an almost total absence of polymorphonuclear leucocytes.

Ætiology.

Although gas gangrene cannot exist without a gas-producing microbe, there are many wounds where the organism is present without any gangrene, and with only a trace of gas infection. These microbes being largely of intestinal origin, are specially associated with the fæcal contamination of skin, clothing, and soil. Their virulence is increased enormously by the presence of streptococci. Although the numbers of wounds of the upper limb and chest are greater than those of the pelvis and lower limb—viz., 299 to 197—yet the mortality is three times as great among the gas infections of the lower limb as among those of the upper, viz., 31 to 10 (including three from tetanus). The nearer the soil the greater are the possibilities of infection.

Projectile.

The general proportion of shell to bullet wounds we have found is as 2 to 1. In cases of gas infection the proportion is as 3 to 1, and in cases of gas gangrene the proportion has been 6 to 1, due probably to the infected piece of clothing which the jagged shell carries in, and the greater destruction of tissue. In nearly every case of severe gas infection, a foully smelling piece of cloth, *capote*, can be found in the wound.

Foreign Body.

Occasionally, more especially after transport, quiescent cases flare up with signs of acute infection. The X-rays show no projectile, but one finds in the depths of the wound a piece of infected *capote*. The infection has probably been kept under control by the use of strong disinfectants, but breaks out when there is a prolonged interval between the dressings.

Interval between Wound and Treatment.

In some of our worst cases of gangrene an interval of several days had elapsed between the infliction of the wound and the first surgical intervention. In contrasting the gravity of these cases nothing has been more remarkable than the influence of this interval, other conditions being equal. After an attack at the end of May, 1915, our cases came in unusually early, indeed after a few hours. The wounds were very similar in character to others coming in on other occasions after a longer lapse of time, but the results were immeasurably superior, and not a case of massive gas gangrene nor a death occurred, although gas infection was prevalent. Whereas when suitable surgical treatment is delayed even a slight flesh wound may be fatal in the presence of a virulent infection, as in the following example:—

Le Soldat J., aged 21, was wounded at midnight on September 13, 1916, by shell in the right triceps about the middle of the arm. He received his first dressing a quarter of an hour later; anti-tetanic serum two hours later, and another dressing in an ambulance the following day, where the shell was localized. On September 16, at 10.45 p.m., the patient was admitted to Royaumont. There was a foul smelling penetrating wound of the right arm, which was enormously swollen from elbow to beyond the shoulder. The skin was tense and white, except round the wound. There was no crepitation. The X-ray plate showed a piece of shell and some deeply seated gas in the muscles. There was no fracture. Temperature 102·2° F., pulse 88. An hour after admission the patient was taken to the theatre, the wound was opened up, and the piece of shell removed with a foul smelling piece of *capote*. The triceps was gangrenous. The provisional bacteriological report was grave, and I decided with considerable reluctance to amputate, and did this well above the wound, by circular method with lateral incisions. All obviously infected deep muscle was cut away. Yellow serosity poured out. The following day the stump was clean, and there was no gas; temperature 99° F., pulse 90. On September 18 gas was seen coming from the wound, and the subcutaneous tissues were œdematous; temperature 101·8° F., pulse 122. At 1.30 p.m. the arm was amputated at the shoulder joint and gangrenous muscle cut away. On September 19 gangrene was still spreading and the wound was very foul. The general condition of the patient became serious. The pulse was rapid and poor and the extremities cold. There was frequent vomiting with dyspnœa. The patient was conscious until death at 8.30 p.m.

This case was a typical one of toxic or œdematous gangrene. In the wound, *Bacillus perfringens*, *Bacillus sporogenes*, and *Bacillus œdematiens* were found, and from the blood the presence of *Bacillus sporogenes* and another unknown organism were reported.

It is quite clear that it is impossible to contrast the results of different antiseptics and dressings unless the interval between wound and operation is considered. I have found that early removal of the foreign body with adequate drainage, cleansing of the wound, and suitable treatment of any accompanying bone or joint lesion has been followed by favourable results whatever exact form of dressing has been applied, unless there is a very virulent infection.

Vascular Lesions.

Vascular lesions have an important influence in the production of gangrene. With complete blockage of the circulation a suitable soil is prepared for gas gangrene, though the gangrene is not necessarily due to the gas infection. It is obvious that such conditions favour the rapid development of gas organisms. Twenty-two cases of gas infections with vascular lesions have been noted, of which six only have been followed by gangrene.

Superficial Femoral Artery.

Case I.—Le Soldat B. Penetrating shell wound of right thigh involving superficial femoral artery with ligature of superficial femoral, four days before admission; large abscess in thigh. *No gangrene.*

Case II.—Le Soldat G. Infected wound of thigh by bullet; superficial femoral ligatured before admission; considerable oozing. *No gangrene.*

Popliteal Artery.

Case III.—Le Soldat C. Infected wound of calf; traumatic aneurysm of posterior tibial; ligature of popliteal; gas gangrene of leg; amputation; septicæmia. Death. *Bacillus perfringens.*

Case IV.—Le Soldat N. Perforating bullet wound of popliteal space with wound of lower part of popliteal artery; traumatic aneurysm; ligature of popliteal artery. *No gangrene.*

Case V.—Le Soldat L. Shell wound of thigh with traumatic aneurysm in popliteal space; anaërobic infection; ligature of popliteal above and below tear of artery. *No gangrene.*

Case VI.—Le Soldat S. D. Traumatic aneurysm of popliteal space by shell; *Bacillus perfringens, Streptococcus;* artery ligatured. *No gangrene.*

Posterior Tibial.

Case VII.—Le Soldat T. Infected shell wound of calf; hæmorrhage from calf; *Bacillus perfringens* infection; ligature of posterior tibial; streptococcal septicæmia; gangrene; amputation. Recovery.

Case VIII.—Le Soldat M. Infected shell wound of calf and knee-joint; secondary hæmorrhage; ligature of posterior tibial. *No gangrene.*

Case IX.—Le Soldat B. Aneurysm of posterior tibial by penetrating infected shell wound; posterior tibial ligatured; *Bacillus perfringens. No gangrene.*

BRACHIAL ARTERY.

Case X.—Le Soldat M. Brachial artery injured by shell; infected wound; *Bacillus perfringens;* ligatured. *No gangrene.*

Case XI.—Le Soldat C. Brachial artery injured; all tissues in antecubital fossa torn across; gangrene; amputation: *Bacillus perfringens.*

Case XII.—Le Soldat G. Extensive infected wound causing thrombosis of brachial; fracture of humerus; amputation; *Bacillus perfringens;* septicæmia. Death.

Case XIII.—Le Soldat A. Secondary hæmorrhage from brachial artery; infected wound; ligature above and below. *No gangrene.*

Case XIV.—Le Soldat C. Perforating wound with thrombosis of brachial artery; ligatured above and below; *Bacillus perfringens. No gangrene.*

ULNAR ARTERY.

Case XV.—Le Soldat E. H. Ulnar artery torn in infected wound; ligatured; drained; followed by gangrene; amputation; cocci; *Bacillus perfringens.*

Case XVI.—Le Soldat Le P. Ulnar artery torn; ligatured in gas infected wound; *Bacillus perfringens, Bacillus sporogenes. No gangrene.*

RADIAL AND ULNAR.

Case XVII.—Le Soldat M. Both arteries torn across by shell; advanced gangrene of forearm; amputation; severe anaërobic infection. Death. *Bacillus perfringens, Bacillus sporogenes, Bacillus histolyticus.*

VENOUS.

Case XVIII: Femoral.—Le Soldat O. Infected wound of thigh, *Bacillus perfringens;* thrombosis. *No gangrene.*

Case XIX: Femoral.—Le Soldat C. Infected wound of thigh; thrombosis developed after gangrene had cleared up; *Bacillus perfringens, Bacillus sporogenes, Bacillus pyocyaneus.*

Case XX: Popliteal.—Le Soldat Q. Infected wound of popliteal space; thrombosis of popliteal vein. *No gangrene. Bacillus perfringens.*

Case XXI: Popliteal.—Le Soldat D. Infected wound of popliteal space with wound of external articular artery and popliteal vein; artery ligatured. *No gangrene.*

Case XXII: Axillary.—Le Soldat M. Infected shell wound with tear of axillary vein and hæmatoma of axilla; *Bacillus perfringens;* ligature of vein. *No gangrene.*

Ligature of an important artery or vein singly does not seem to increase the incidence of gas gangrene noticeably. Wounds which affect both venous and arterial channels are more serious. In two cases of ligature of the superficial femoral artery, and in two of thrombosis of the femoral vein no gangrene occurred. In four cases of ligature of the popliteal artery gangrene occurred in one only. In three cases of ligature of the brachial artery, no gangrene occurred, in one where both artery and vein were injured there was gangrene; in another thrombosis of the artery occurred as a secondary result of the infection.

In the following case of wound of the brachial artery the gangrene was partly due to the obstruction to the circulation. The case was of a mild type. There was no gas infiltration nor œdema of the subcutaneous tissues of the arm itself. It was possible to do a flap operation with the expectation of getting a good result.

Le Soldat C., aged 42, wounded on July 1, at 10 p.m.; perforating wound of the left arm and forearm, with dirty, ragged edges exposing the gangrenous extensor carpi ulnaris, supinator longus, extensor carpi radialis and biceps. The brachial artery was divided and thrombosed. The hand and forearm were blue and swollen; no radial pulse could be felt. Gas in the wound. Temperature 102·6° F., pulse 102. Good general condition. July 2: The left arm was amputated by circular method, with short flaps about the middle of the arm. X-ray: No fracture or foreign body. Some irregular gas tracery, *Bacillus perfringens* and other anaërobes present. Evacuated, healing on July 14, 1916.

Ligature of Popliteal Artery.—Le Soldat La R. on July 1 received a perforating grenade wound of the right thigh, with wound of the popliteal artery and formation of a traumatic aneurysm, and a perforating anaërobically infected wound of the right calf. On July 3, the artery was ligatured immediately below Hunter's canal, and the clot cleaned out. Through drainage was established in the calf and free lateral incisions made. Although very considerable swelling followed, the foot remained warm and pulsation returned. The patient made a good recovery.

Ligature of Posterior Tibial Artery.—Le Soldat M. had penetrating wounds of the left knee-joint and calf with severe anaërobic infection. The joint was washed out and drained down to the synovial membrane, a piece of shell which had fractured the patella and penetrated the internal condyle being left. Secondary hæmorrhage occurred twice from the calf wound which had been drained and opened up widely. After a third more severe

hæmorrhage the posterior tibial artery was ligatured. The patient made a goodrecovery. The piece of shell was extracted later with aseptic precautions and the patient got back very good movement in the knee-joint.

Thrombosis of Poptiteal Vein.—Le Soldat Q., aged 41, received a penetrating shell wound of the right calf extending into the popliteal space, on September 12. He was admitted at midnight on September 18; temperature 102·6° F., pulse 104; blanched but not collapsed. The right leg and foot were swollen but warm and not discoloured, and the anterior tibial pulse was felt. There was a diffuse swelling in the popliteal space. The X-ray plate showed a large piece of shell lying behind the femur, and gas in an abscess cavity round it. There was no fracture. The popliteal space was very freely opened up and the calf around drained, pus evacuated and the projectile removed. Part of semi-membranosus muscle was grangrenous and was cut away. The popliteal vein was thrombosed. The leg was put up partially flexed on the thigh with a continuous saline irrigation through the wound. The tube (a small one) was removed in four days. The wound rapidly cleaned, the swelling of the leg disappeared and recovery was rapid. Bacteriological examination of the pus and shell showed cocci and *Bacillus perfringens*.

Wound of Axillary Vein.—Le Soldat M. received a penetrating shell wound of the left axilla. On admission the axilla was full of clot and a large piece of infected shell lay under the pectoral muscle. On opening up the axilla and cleaning out the clot there was free hæmorrhage from a lesion of the axillary vein. This was ligatured on each side. The artery was uninjured. The case cleared up well.

Thrombosis of Brachial Artery.—Le Soldat C. received a perforating bullet wound of the right arm causing division and thrombosis of the brachial artery—no radial pulse was felt. The arm was swollen. Incisions were made over the wound, and clot and hæmorrhagic discharge containing *Bacillus perfringens* evacuated. The artery was tied above and below the thrombosed area. No gangrene occurred.

Thrombosis of Femoral Vein.—Le Soldat A. received a perforating shell wound of the right thigh, with a grazed fracture of the femur, on July 1. Pus and gas exuded from the small exit wound. On July 2 the wounds were incised and gas and pus evacuated. Very open through drainage was established. Bacteriological report showed numerous large sporing bacilli. The wound healed well. Signs of thrombosis of the right femoral vein supervened on July 15, with pulmonary emboli on August 1 and 3. On October 30, the leg and thigh were still considerably swollen, but the patient was otherwise in good condition.

In the following case it is probable that infection had become generalized before the popliteal artery was ligatured.

Le Soldat C. was wounded by a piece of shell in the left calf on October 12. The wound was small and the piece of shell lay behind the tibia. There was some swelling of the calf. On October 26, the shell was

removed, pus evacuated, and free through drainage established. On October 27, there was a large swelling of the calf, which was found to be due to hæmorrhage from the posterior tibial artery. On account of the swelling of the leg it was found impossible to tie locally, and the popliteal artery was ligatured. On October 28, the foot was cold, there was no pulse, and crackling was present in the calf. Amputation was performed in the middle of the thigh, in which there was some gas formation. On October 29, although the stump looked clean the general condition of the patient was bad; temperature subnormal, pulse 130, and respiration rapid. The skin became increasingly yellow. On October 30, there was enormous distension of the stump, a most offensive smell, and gas was discharged from the wounds audibly in reports. There was dyspnœa and hæmorrhagic vomiting. The patient was fully conscious, said he had known from the first that he was fatally injured, but ascribed his bad condition to the fact that he had been gassed some time before and that it had left his chest weak. *Bacillus perfringens*, streptococci and staphylococci were reported. This case clearly had become septicæmic.

In cases of traumatic aneurysm occurring in these infected cases, the absence of gangrene has been noticeable, and the cases I have just described prove that ligature of important vessels is not necessarily followed by gangrene.

Fractures.

The complication of the wound by a bone lesion increases the incidence of gangrene. In 464 cases infected by gas organisms, at least 60 per cent. were complicated by fractures.

Seventy-seven Cases of Gas Gangrene Complicated by Fractures.

Fractures	Fatal	Recovery
Shoulder-joint	0	2
Humerus	2	4
Elbow-joint	0	5
Radius and ulna	1	6
Radius	0	2
Ulna	0	3
Carpus	0	2
Metacarpals	0	6
Ribs	1	1
Vertebræ	0	1
Pelvis and tibia	0	1
Pelvis	4	0
Hip-joint	1	1
Neck of femur	1	1
Upper third of femur	0	1
Middle third of femur	1	0
Tibia and fibula	1	7
Fibula	3	3
Tibia	1	4
Tarsus	0	6
Metatarsal and multiple wounds	1	2
Phalanges	0	1
Jaw with multiple wounds	1	0
	18	59

In 107 cases of gas gangrene, 71 per cent. had fractures; in twenty-five fatal cases 72 per cent. had fractures. It will be seen from the accompanying table that fractures of the pelvis, the hip-joint and upper end of the femur are extremely fatal. They are frequently associated with wounds of the bladder and rectum.

Situation of the Wound.

Wounds of the calf involving the vessels, with or without fracture, are dangerous unless treated early and energetically. There is a tendency to the accumulation of gas and pus under the deep fascia and to increased absorption and spread of infection. In the same way wounds implicating the trunk, hip-joint, or pelvis are grave because the opportunities for free drainage are limited. Even a mild gas infection may lead to a fatal termination in such a situation.

Le Soldat C., aged 20, wounded on July 8, was admitted on July 11 with a perforating bullet wound of the right hip. The general condition was excellent; temperature 98° F., pulse 72. The first X-ray photograph was imperfect, but in the absence of symptoms and with a favourable bacteriological report, conservative treatment was adopted. On July 13, as the temperature and pulse steadily rose, the patient was examined under chloroform and a fracture of the hip-joint determined. A further photograph showed comminution of the neck of the femur. Sequestra were removed and a drainage tube passed through. Bacteriological examination of a sequestrum showed very scanty *Bacillus perfringens*. Two days later gas was seen to be exuding from the wound and the temperature was 102·6° F., pulse 104. There was no offensive smell. The head of the femur was excised and the wound very widely opened up. Swelling of the muscles still progressed, and on July 16 I amputated at the hip-joint. The patient rallied, but the yellow tint of septicæmia developed. The stump continued to swell, pouring out hæmorrhagic discharge, and death ensued the following day. The patient was fully conscious to the end.

Tissue Injury.

Tissue injury is one of the most important factors in gas gangrene and partly accounts for the gravity of shell as compared with bullet wounds. This influence can be seen when even a small injury, such as a subcutaneous injection, causes the formation of a gas abscess in a septicæmic patient.

Increased Intramuscular Tension.

Increased intramuscular tension is a powerful factor in the transition of a gas infection to gas gangrene. Where the wound of entry is small

or closed the bacterial products accumulate under tension. The muscles involved tend to become gangrenous early from arrested circulation due both to pressure and absorption of toxin. When the sheath bursts gas spreads very rapidly in the subcutaneous tissues.

Pressure from Outside.

Tight bandaging, and more especially plaster of Paris splints, act in a similar manner. Some of the worst cases of gangrene have arrived with the fractured limb encased in plaster, with the swollen tissues bulging above and below. The French *gouttières* are not attended by this danger, and for transport of freshly wounded soldiers are very useful and comfortable. The one flaw in the favourite Thomas's splint lies in the pressure from the shoulder or thigh ring.

Joint Injuries.

Joint injuries, while considerably increasing the gravity of the case do not seem to have any direct influence in the production of gas gangrene. Among cases of massive gangrene a joint lesion was present in under 20 per cent.—sacroiliac, 1; hip, 4; knee, 4; ankle, 2; tarsus, 4; shoulder, 2; elbow, 4; wrist, 4. In the cases of gas infection a joint was implicated in 13 per cent.—hip, 6; knee, 9; wrist, 10; ankle, 3; mid-tarsal, 8; shoulder, 7; elbow, 19. The necessity for immobilization with joint infections is unfavourable, as it is by no means easy to apply a fixative apparatus on a restless patient without a certain amount of bandaging and pressure. This is shown in two cases of elbow-joint gas infection in a Senegalese and Arab, respectively, where amputation became necessary. Under ordinary circumstances these would undoubtedly have yielded to resection or drainage, if the joint could have been kept quiet without much bandaging. Enforced action after a joint injury leads to the spread of infection and to pyemia or septicæmia.

Le Sergent T., after being wounded on September 25, in the left knee-joint with fracture of the patella, was obliged to walk 500 metres. On admission on September 27, the knee-joint was filled with infected, blood-stained, synovial fluid. Infected *capote* and splinters of bone were removed, and the joint washed out and a drain inserted down to the joint on each side, and the joint immobilized. Symptoms of pyæmia with infections of other joints rapidly supervened.

This case was noteworthy, as it was the only death which occurred in a series of thirty cases of wound infection of the knee-joint.

Fatigue.

How much influence muscle fatigue has on the incidence of gas gangrene remains to be shown, but it is noticeable that the majority of cases occurred after an attack. It is possible that the formation of certain products of muscular activity may lessen this resisting power of the tissues to bacterial infection—more especially to gas infection which attacks muscle.

Bacteriology.

As I have already stated, owing to the large number of gas cases coming in together, it was not always possible to have every case fully studied. In addition, however, to our own bacteriologists, Dr. Weinberg, of the Pasteur Institute, and his colleague M. Séguin, were good enough to study in detail the flora of a considerable number of cases, and from their expert knowledge of this very special branch of bacteriology, were able to give us, not only information of the greatest value, but also serum specially prepared for some of the different forms of anaërobic organisms, to the employment of which I shall refer later.

Some interesting facts stood out. First, the multiplicity of organisms infecting a wound. Secondly, the preponderating presence of *Bacillus perfringens*, which was found in practically every case of gas infection. When occurring alone it did not appear to be very dangerous unless deeply seated so that drainage could not be freely established. It did not invariably produce gas or a specially foul smell in the wound. Frequently from its hæmolytic properties, or action on capillary walls, there was reddish discharge, together with discoloration and bronzing of the skin, in addition to gas formation. In the X-ray photograph swelling of tissue only was seen generally. Occasionally in the early stages of gas formation a dappling of gas bubbles. The only deaths occurring where *Bacillus perfringens* alone was reported to be present were fractures of the pelvis and thorax.

Bacillus sporogenes was reported in culture in forty-one cases and it was noticeable that, although there was an excessive production of gas and a foul smell, the cases generally yielded to treatment unless combined with more virulent organisms. *Bacillus sporogenes* often lingers when other organisms die out. The presence of *Vibrion septique* was reported six times, all very severe cases. In three cases Dr. Dalyell found *Bacillus Hibler IX.*[1] Some of the more newly described

[1] Robertson, *Journ. Path. and Bact.*, January, 1916, p. 333.

organisms such as *Bacillus histolyticus*,[1] and *Bacillus œdematiens* (fifteen cases) were also reported. Again and again it was noted that the association of the streptococcus (fifty-nine cases) with the anaërobes increased the severity and gravity of the infection, a considerable number of cases dying with streptococcal septicæmia. While the anaërobes attack the muscle, the streptococcus spreads in the fascial planes. In many cases where the surface wound showed only aërobic infection anaërobes could be grown from the deeper tissues or sequestra. Negative bacteriological reports must be received with a certain amount of reserve on this account.

Tetanus.

In fifteen cases there was clinical (eight) or bacteriological (seven) evidence of the presence of tetanus.

Three of these were local, with symptoms starting in one limb but becoming generalized. Symptoms occasionally developed weeks after the original injury, in one case when the wound had healed. Owing to the uniform early administration of antitetanic serum, the symptoms were masked and their very vague character made the diagnosis extremely difficult. This is peculiarly unfortunate as in such cases an early diagnosis is followed by good results if massive doses of serum are administered intrathecally, or, failing this, subcutaneously. The following is such a case:—

Le Soldat M., aged 24, wounded on July 4, 1916, at 1.30 p.m. by shell, was dressed immediately and given an injection of antitetanic serum the following day. On admission there were multiple muscular wounds of the arms, legs and left thigh, with anaërobic infection of the right leg and right thigh. On July 6 three pieces of shell were extracted. The wounds healed quickly, but the patient was sleepless and became rather disagreeable in temper. On July 18 there was pain in the muscles of the right calf and right foot, but no spasmodic movements. July 20: Some pain in the right calf and heel. On July 29, a piece of shell was removed from the adductor longus. Bacteriological report, sterile. After operation the pain in the foot was less for a few days, but the patient still slept extremely badly, even after morphia. On August 11 there was recurrence of pain in the right foot and calf and little jerks of the foot when the patient tried to straighten out the knee, which was rigid; characteristic facies (a mild form of risus sardonicus) giving a peculiar expression and very worried look; 10 c.c. antitetanic serum injected subcutaneously; bromide of potassium and hydrate of chloral were given three times

[1] Weinberg et Séguin, "Contributions à l'étiologie de la gangrène gazeuse." *Comptes Rend. de l'Acad. des Sciences*, 1916, clxiii, p. 449.

a day. August 12: No spasms but rigidity of the calf muscles continued; 30 c.c. antitetanic serum at 3 and 7; in the evening no rigidity of the calf. August 13: Hyperæsthesia of the calf muscles, stiffness of the knee; 30 c.c. antitetanic serum 7 p.m. August 14: No involuntary movements, slept better; 30 c.c. antitetanic serum at 3 p.m. and 7 p.m. August 15: Rigidity of the right knee; pains in the arms; 30 c.c. antitetanic serum at 10 a.m., 2 p.m. and 6 p.m.; slept; says that tight feeling in leg decreased. August 16: No pain in arm and less in leg, which can be extended; tingling in right foot and pain in sole of foot. August 17: Twitching of the face muscles and rigidity of knee; lumbar puncture: 30 c.c. cerebrospinal fluid extracted and the same quantity injected, antitetanic serum, saline subcutaneously, Oij; four hours later temperature 105·8° F.; pulse 118; headache; cold sponging; mag. sulph. 5ii and saline 20 oz. *per rectum;* temperature fell to 100·4° F.; pulse 100; no pain in toes or twitching of face; slept well with morphia; some sweating. August 20, 8 a.m.: Head retraction, twitching of face and body for two minutes; very excitable and talkative; pain and rigidity in both arms, none in legs; again 30 c.c. cerebrospinal fluid withdrawn by lumbar puncture (slightly turbid); 25 c.c. antitetanic serum injected; quiet during day, some twitching of face; temperature 104° F. at 2 p.m. August 21: Very restless; morphia $\frac{1}{4}$ gr. at 9.30 p.m. and $\frac{1}{6}$ gr. at 2 a.m.; during day twitching of face, pain in arms, restlessness; complains of sore throat; no rigidity. August 22: Pain in neck muscles, twitching of face, pain in arms, slight attack of bronchitis. August 23: Improvement, but some twitching of face. August 24: Some twitching of face; no further facial spasms, and gradual improvement. October 10: Still walks badly; electrical reaction shows response of all the leg muscles, sluggish. October 13: 5 c.c. antitetanic serum given. October 14: 10 c.c. antitetanic serum. October 15: 10 c.c. antitetanic serum. October 16: 20 c.c. antitetanic serum. October 17: Shell localized in line of right sciatic nerve and removed from surface of nerve; nerve nodular and fibrous; wound drained; 30 c.c. antitetanic serum given subcutaneously at operation. October 18: Pain in thigh but none in foot; knee flexed, cannot be extended; some twitching of face; 40 c.c. antitetanic serum subcutaneously. October 23: Wounds healed. The patient began to improve very much after this operation and walked with only a slight limp when he left the hospital early in November. His facial expression had become normal, he was sleeping well, and was no longer irritable.

The most characteristic feature about these obscure cases of delayed, latent, or masked tetanus is the peculiar facies. They have an anxious, worried look, which appears usually a little before or coincidently with the pain in the affected limb. They do not sleep well, and careful examination may discover muscular rigidity. The administration of serum by means of lumbar puncture has been infinitely more successful than any other method of treatment.

Clinical Forms.

Cases of gas gangrene and gas infection group themselves into several different classes.[1]

(1) The classic form associated with much gas production and little œdema.

(2) The toxic, œdematous form, where œdema is the most striking feature, though gas may be present.[2]

(3) A mixed form, associated with both gas production and œdema.

(4) Local gas abscess.

(5) Superficial and deep-seated gas phlegmon.

(6) Chronic and latent infection.

(7) Septicæmia; pyæmia.

(1) In the *classic* well-known form there is a darkish red discharge from the wound, a putrefactive odour, great swelling of the limb, crepitation, and the formation of hæmorrhagic blebs with bronzing and discoloration of the skin. Nearer the trunk the superficial veins are distended, and a yellowish œdema surrounds them. With the onset of septicæmia, as the case progresses to a fatal conclusion, there is often a lemon yellow coloration of the skin, an increase in the pulse-rate, sweating, coldness of the extremities, subnormal temperature, hæmorrhagic vomiting and dyspnœa. The patient talks naturally, may eat well, and is generally conscious almost to the end. The following is a typical case :—

Le Soldat B., aged 20, received a perforating wound of the right leg and left foot on July 16 at noon (*see* fig. 5, p. 69). He was dressed the same day, a dry dressing being put on the right leg and a tube through the left foot. At midnight on July 17, thirty-six hours later, he was admitted to Royaumont. The patient was restless and talkative. He was pale, with a slight flush over the cheeks. The pupils were widely dilated. Temperature 102° F., pulse 128, weak but regular. The right leg was swollen and discoloured up to the knee and the right foot was cold and gangrenous. Over the outer side of the middle third of the calf was a small dry wound. Above it were bullæ distended with dark fluid. There was blueness and discoloration at the upper and back part of the calf. From the knee to Poupart's ligament the thigh was of normal colour, but swollen and distended with gas. There was very marked crepitation. No swelling nor discoloration on the abdomen. In the left foot was a perforating

[1] I agree with Dr. Weinberg's classification into classic and toxic forms, "Bacteriological and Experimental Researches in Gas Gangrene," *Proc. Roy. Soc. Med.*, 1916, ix, (Occ. Lect.), p. 120.

[2] This form closely resembles cases described by Major Hull as white gangrene. *Brit. Med. Journ.*, May 27, 1916, p. 756.

unhealthy wound at the back of the big toe—no foul smell. The patient was immediately given morphia $\frac{1}{4}$ gr., with atropine $\frac{1}{100}$ gr., and a rectal saline, which was retained. The general condition was very bad still, but at 3 a.m., July 18, the thigh was amputated through the upper third of the femur. There was an enormous amount of gas in the muscles, so that when it escaped the tourniquet relaxed. There was no hæmorrhage, as the artery was controlled digitally. At the end of the operation the patient was almost pulseless. A rectal saline with brandy was administered, also strychnine hypodermically. When put back to bed he was restless, with profuse perspiration. The pulse was weak, the lips were pale, the colour greyer than before. During the day there was some improvement. The pulse was rather better and the stump looked fairly clean. There was no obvious gas either in the stump or abdomen. The left foot, however, became gangrenous. There was no swelling of the ankle, but mottling of the skin up the calf. An amputation was impossible. On July 19, after a quiet night at 6 a.m., the pulse became weak and the patient restless. The colour of the skin became extremely yellow. Death took place at 9.30 a.m.

In the wound *Bacillus perfringens* and *Bacillus histolyticus*, and in the blood culture *Bacillus perfringens* were found. The X-ray photograph shows a remarkable condition of the muscles of the right knee and leg. This patient was already septicæmic on admission and probably hopeless from the first.

In another less typical case of classic gas gangrene, *Vibrion septique* was found in the wound and blood, but the patient died before the suitable serum could be administered in quantity.

A. S., a young Senegalese, was admitted on July 23, at 1 p.m., having been wounded three days previously in the right leg and jaw. The wounds had already been treated in an ambulance. There was a comminuted fracture of the right fibula and the leg was cold, gangrenous, and distended by gas as far as the knee-joint. Temperature 100° F., pulse 120. A circular amputation with lateral incisions through the upper third of the thigh was performed almost immediately and a piece of shell extracted from the lower jaw. Immediately after amputation, before the bacteriological diagnosis was made, 72 c.c. mixed serum were injected. The X-ray plate showed much streaking and linear separation of the muscle fibres by gas. The following day the patient seemed well, the stump was clean and not swollen until the afternoon, when it became enormously distended. *Vibrion septique* was reported in the wound and blood. At 6 p.m., after sitting up and talking rapidly, the patient suddenly fell back unconscious, and died shortly after. This sudden termination suggested gas embolism.

This organism, *Vibrion septique*, has been reported in only a few of our cases, but these have nearly all been fatal or, if recovery has taken place, it has been after amputation wide of the affected region.

(2) *Toxic or Œdematous Form.*—This form of gas gangrene, though relatively infrequent, is very fatal. The limb swells rapidly, but there is no obvious gas formation nor crepitation except in the deep muscles. The limb is firm with solid œdema, and the skin pale. The engorged superficial veins stand out. The following is a typical example :—

Le Soldat D., aged 30, wounded on August 1, at 3 a.m., and admitted on August 5 with comminuted fracture of the lower end of the ulna and radius caused by a perforating piece of shell. The wound had been drained on August 2 and a considerable amount of muscle had been cut away. On admission, temperature 100·8° F., pulse 100. The wound looked very dirty. It was cleaned under an anæsthetic, and some sequestra removed. There was a slight amount of yellow œdema round the wound which suggested anaërobic infection, but the first cultures, owing probably to the strong disinfectants, of which the wound still smelt, were negative. The same evening 25 c.c. anti-*œdematiens* serum were given subcutaneously. The next day, August 6, the wound looked healthy ; it was dressed twice. On August 7 the hand was puffy, and two hours later there was swelling to the elbow-joint. The wound was extremely offensive, and the muscles of the lower portion of the forearm gangrenous. The forearm was amputated, and incisions into the subcutaneous tissue were made and much yellow serum drained away. The ends of the fractured ulna were bathed in foul pus, and the radio-ulnar joint acutely inflamed. Bacteriologically the pus showed on culture a large aërobic spore-forming bacillus, a streptococcus and an anaërobic organism presenting the characters of *Bacillus œdematiens*. The blood culture was negative. The same evening 25 c.c. anti-*œdematiens* serum were given subcutaneously. The wound looked healthy until August 10, when at 2 p.m. (temperature 101·2° F., pulse 88) the flaps and tissues began to swell rapidly. The stump was very dirty and offensive. At 3 p.m. amputation was done through the lower third of the arm ; 40 c.c. of anti-*œdematiens* serum was given. Progress was uneventful until August 16, when there was more swelling. The stump was pale and doughy. There was no crepitation, but the superficial veins were prominent ; 40 c.c. anti-*œdematiens* and 30 c.c. antistreptococcal serum were given. Later in the day the swelling increased, and there was œdema extending up into the axilla, especially along the course of the artery. A further circular amputation high up in the arm was done with lateral incisions. There was a considerable amount of œdema along the course of the vessels, which were tied as high as possible, but no clot was seen in the vein. The bone marrow was curetted out, and the cavity packed with gauze swabs and iodine (1 in 1,000). Good progress was made. The bacteriological report indicated the presence of *Bacillus Hibler IX* and *Bacillus œdematiens*. On November 10 reamputation with flaps. Good recovery.

In this form of gas gangrene the projectile is usually seated in the deeper muscles, in which gas formation takes place under tension.

There is great tendency to toxæmia from absorption of the products of these organisms, with thrombosis of the deep veins, and resulting œdema.

(3) *Mixed Forms, showing Œdema and Gas Production.*—In this variety, which is very common, there is both gas formation and œdema in varying proportions.

Le Soldat F., aged 21, was wounded on July 8, 1916, in the right thigh by a shell, and by a bullet in the left foot, fracturing all the metatarsals. He was admitted on July 10 with an extensive septic infected wound of the external and posterior surface of the middle third of the thigh, involving mainly the vastus externus. There was no fracture. The smell was extremely foul. The patient was brought up to the theatre, and a piece of shell removed and much gangrenous muscle cut away. Amputation was not considered necessary as there was no fracture and the wound was apparently superficial. On July 11 the wound still looked dirty, but nothing special was noted. July 12: Early the following morning when the wound was dressed the patient complained of uneasiness in the leg, which was still very dirty but not more swollen. At 2 p.m. he began to cry out with pain. On examination a blue discoloration of the skin was seen above and below, and there was swelling of the thigh above the wound. At 2.15 p.m. a circular amputation of the thigh in the upper third was done. The wound extended as high as the lesser trochanter, and gas exuded from the muscles. Incisions were made in the œdematous cellular tissue. The patient was very much collapsed. Intravenous and subcutaneous salines were given. The pulse was uncountable, the temperature subnormal and discoloration and gas formation appeared at the site of subcutaneous saline injections. Death took place twelve hours later. The blood culture (July 12) showed the presence of *Vibrion septique* and *Bacillus œdematiens.*

In this case had it been possible to diagnose the presence of *Vibrion septique* earlier, amputation might have yielded a more favourable result, though, owing to the situation of the wound, it would have been impossible to amputate through healthy tissues.

(4) *Localized Gas Formation, or Gas Abscess.*—The following is an illustrative case:—

Le Soldat A., aged 40, wounded on August 22 by a penetrating shell-wound in the muscles of the right scapular region, was admitted on August 26. A large piece of shell was present, and a resonant swelling over the point of localization, with spreading œdema round it. On incision, a large amount of gas and pus escaped with the projectile, from which *Bacillus perfringens*, streptococcus and *Staphylococcus aureus* were reported. On September 9 a further incision was necessary as the temperature was 103° F., and a streptococcal infection with abscess formation lower down in the back developed.

This case is typical of the course of events with superimposed streptococcal infection. The gas infection subsides, but the streptococcus is resistant to treatment and resembles in behaviour the most virulent intestinal strains. The presence of the streptococcus, in addition to a gas organism, can often be suspected when there is both gas in the muscles and reddish-yellow pus formation along the fascial planes, with the typical rose infection of the skin. Even with a superficial lesion the combination is dangerous, but in deep-seated cases it is very fatal, from the tendency to streptococcal septicæmia.

(5) *Gas Phlegmon.*—This class comprises at least four-fifths of anaërobic wound infections. It may be superficial (in the cellular tissue) or deep (in the muscles). Although gas is present in the wound the inflammatory process is localized and does not often extend to gangrene, but yields to incisions and drainage. The muscles are swollen and friable; there is much yellow œdema in the subcutaneous tissues. The following is an illustrative case:—

Case J. (*see* fig. 11, p. 75).—Le Soldat P., aged 24, wounded in the right forearm by an explosive bullet with comminuted fracture of the ulna on July 1, at 10 a.m. Admitted the following afternoon, temperature 97° F., pulse 100. Very exhausted. Skin sallow. Large foul-smelling wound on ulnar border of right forearm, with muscles and tendons cut. A small punctate entry wound on the anterior aspect of the wrist in the midline with the whole forearm swollen and baggy. Operation on July 2, the lower entry wound enlarged upwards, and pieces of loose bone removed from both wounds. Gas bubbles and reddish discharge from the wound. Incisions through deep fascia for relief of tension and drainage. Put up supinated on back splint. July 4: Arm slightly less swollen, but wound very offensive. Evacuated September 24, with almost normal movement. Bacteriologically: A severe infection with *Bacillus perfringens*.

Untreated, the condition of gas phlegmon rapidly takes on a more serious form. The bacterial products, accumulating under tension in the muscle sheaths, stop the circulation and gangrene occurs. Gas escapes from the ruptured sheath, blood from the infected vessels with the formation of subcutaneous crepitation and discoloration of the skin, until the ordinary clinical picture of gas gangrene is produced.

(6) *Delayed and Latent Gas Infection: Delayed Form.*—In this form of gas infection the infecting organisms are inactive for a time, and then suddenly take on increased growth and virulence. The following is an instance —

Le Soldat M., aged 44, was wounded in the left leg on October 7, at 12 midnight, by a shrapnel bullet which was removed before admission, and

antitetanic serum was administered as usual. On admission on October 16 there were large, dirty, foul-smelling wounds on the anterior and posterior aspects of the left leg with comminuted fracture of the tibia at the junction of the lower and middle thirds. Much bone had been removed. There was some discoloration over the front of the tibia. The leg was in plaster on arrival. The wounds were thoroughly cleansed with salt and carbolic, and packed with salt tablets. The following day continuous eusol irrigation was begun. The wound still looked extremely foul. October 18: *Bacillus tetani*, *Bacillus perfringens*, *Vibrion septique*, streptococcus, and *Bacillus proteus* reported. On October 26, nearly three weeks after the injury, the temperature rose to 103° F., and pulse 128, at 8 p.m. The leg was rather more swollen, and there was a suspicion of crackling in the skin. At midnight the pulse was 132, temperature 104° F., tongue dry and brown. I amputated, half an hour later, above the knee-joint, by open circular method. The patient did well, although there was considerable inflammatory reaction in the stump with abundant *Bacillus perfringens*, streptococci, and coliform bacilli. No signs of tetanus developed, and the temperature and pulse came down after amputation.

The X-ray photograph shows the appearances of gas ten days before operation.

Latent Form.—Anaërobic organisms may remain latent many months round a piece of shell or *capote*—hence such cases can in no sense be regarded as clean operations, for there is every probability that if treated as such, acute sepsis will supervene. At any time they may light up and cause abscesses, pyæmia, or septicæmia. If drained carefully they usually do well, but an inflammatory exacerbation must be expected. This makes brain and joint explorations for latent projectiles peculiarly dangerous. In bone, particularly, do the organisms lurk, hence the necessity for draining re-amputations, even when a careful bacteriological examination of the surface wound gives a negative report. Often the anaërobic gas organism dies off but the streptococcus remains, while latent cases of tetanus are by no means uncommon. A typical case of latent wound infection is the following:—

Case L. (*see* fig. 12, p. 76).—Le Sergent V., aged 23, was wounded on February 8 at 3 p.m., by shell and he had a penetrating wound of the left knee-joint, and a perforating wound of the right eyeball, with penetration and fracture of the ethmoid. On admission on February 12: Temperature 98·4° F., pulse 60. The right eye was disorganized. On February 14, it was enucleated, the cavity packed with salt and carbolic gauze. The foreign body was not looked for, as it had not been possible to localize it. The wound in the left knee-joint was explored. The piece of shell was found at the end of the sinus, from which dark red discharge exuded. February 16: Temperature 103·8° F., pulse 100; severe headache; restlessness; continuous boracic irrigation. February

18: Delirium; some blood-stained discharge from the right nostril. February 19: Temperature still high; eye condition improving, but swelling of knee-joint; on aspiration, thin, flaky pus containing streptococci was withdrawn; the capsule was incised by lateral patellar incisions, the joint washed out with hot salt and carbolic lotion and drained down to, but not into, the joint. A Thomas's splint was applied. March 3 and March 11: Further incisions into joint above patella. April 19: Wounds nearly healed and good movement obtained. May 4: The patient still complained of headaches and a sensation of heaviness in the back of the head. June 2: After localization the foreign body was removed by way of the empty orbit, from the right middle ethmoidal cells, through the fractured ethmoidal plate, with the finger, after direct examination under X-rays. There was considerable hæmorrhage from nose and throat. (I now use the Baume-Pluvinel *doigtier*, which is a modified but much more powerful form of telephone probe.) Pus was freely discharged from the sinus for some days. The patient was sent, on June 19, to an ophthalmological centre, for a false eye. Knee movements perfect. Bacteriological report on piece of shell removed from ethmoid: Smears show thick purulent material lying in irregularities with granular amorphous material, and pus cells containing streptococci in chains. On culture a good growth of streptococci in long chains and a few *Bacillus perfringens*. June 5: Streptococci still abundant in pus and *Bacillus perfringens* present in culture.

In this case an interval of nearly four months had elapsed between the receipt of the wound and the removal of the projectile. Encapsuled streptococci are frequently found in the projectiles that have been quiescent some time. In one case of penetrating shell wound of the knee-joint the shell showed streptococci after an interval of a year's duration. In a case in which the shell had reached the peritoneal cavity from a badly-infected buttock wound the projectile was removed through the abdomen after six months from the left side of the rectum with drainage. Encapsuled streptococci were found in the pus surrounding the shell. The patient made an excellent recovery. In a case of non-union of the radius in a Senegalese, which had been regarded as a clean case, healing rapidly, I cut down two months later on the radius, and finding only some fibrous tissue between the two fragments, I removed it and freshened the ends of the bone, closing the wound. The following day the pulse rose and the arm swelled nearly to the shoulder. It was necessary to open up very thoroughly and apply a saline drip. *Bacillus perfringens* was isolated. Good union finally took place.

(7) *Septicæmia and Pyæmia.*—In the great majority of cases an anaërobic infection of the blood is fatal, and occurs as a terminal phase; the temperature falls, the pulse rate rises. A blood culture was made in forty-nine cases, and positive results were obtained in twenty. Of

these thirteen were gas-forming organisms and seven pyogenic, six streptococcus and one pyocyaneus, but double or triple infections were occasionally present. *Vibrion septique*, three; *Bacillus œdematiens*, three; *Bacillus perfringens*, two; *Bacillus sporogenes*, one; *Bacillus histolyticus*, two; unidentified anaërobe, two. In all but two cases of gas septicæmia there was a fatal issue, one recovering after amputation, the other after amputation and serum treatment. The first case was an infection by *Bacillus œdematiens*.

Case I.—Le Soldat R., aged 20, was wounded on July 9, at 2 a.m., and was admitted on July 9 at midnight. The patient was very ill. Temperature 99·4° F., pulse 136, collapsed, the skin yellow, and vomiting was present. There was a perforating, gaping, foul smelling, shell wound of the right forearm, with gangrene of the muscles and fracture of both bones. No X-ray photograph was taken as the patient was so ill. There was every appearance of septicæmia. The patient was allowed to rest, rectal salines and stimulants were given, and operation was delayed for a few hours. The next morning the condition was better; pulse 120. The right arm was amputated through the lower third by circular method. On September 6, re-amputation was done by flaps, but owing to general symptoms of streptococcal infection developing, the wound had to be opened up and continuous irrigation applied. By October 8 the wound had nearly healed. *Bacillus sporogenes* and *Bacillus œdematiens* were reported from the wound and *Bacillus œdematiens* from the blood.

The blood infection must have been a very transient one, as the temperature dropped immediately after amputation.

Case II was specially interesting from the fact that both *Bacillus perfringens* and *Bacillus histolyticus* were recovered from the blood, together with a non-pathogenic anaërobe not yet completely studied, but that in spite of this fact recovery took place. *Bacillus perfringens* disappeared from the blood after the first two injections of serum.

Case II.—Le Soldat G., aged 21, wounded on July 19 by shell, was not dressed for twelve hours, when the left leg and right knee were put in plaster. Admitted on July 21; temperature 102·2° F., pulse 130. In the left leg there were perforating wounds. The skin was discoloured over the anterior border of the tibia; the fractured ends could be seen in the wound. There was swelling of the thigh and prominence of the veins. Over the right knee there were penetrating wounds. Operation at 9 p.m. X-ray examination showed a fractured left tibia and fibula. The thigh was amputated in the lower third. The smell was foul. A large, dirty piece of shell with adherent *capote* was removed from the head of the right tibia. After operation, temperature 98° F., pulse 130. The patient was very ill during the night following operation. July 22, 10 a.m.: 25 c.c. anti-*perfringens* serum and 20 c.c. anti-*œdematiens* serum administered subcutaneously. July 23: 32 c.c. anti-*perfringens* serum

and anti-*œdematiens* serum administered subcutaneously; 75 c.c. anti-*Vibrion septique* serum, anti-*œdematiens* serum, and anti-*perfringens* serum, round knee-joint. July 24: 40 c.c. anti-*perfringens* serum, and 70 c.c. anti-*œdematiens* serum, subcutaneously, and 30 c.c. (mixed) into wound. July 25: Right knee-joint swollen; 2 p.m., temperature 103° F., pulse 102; large amount of gelatinous material and yellow fluid evacuated; drained to synovial membrane; long incision along int. saphena which was distended and surrounded by yellow serosity; the muscles were gelatinous; pulse very poor; 1 pint saline given intravenously; 8 p.m., 40 c.c. anti-*perfringens* serum subcutaneously; 12 midnight, 60 c.c. anti-*perfringens* serum subcutaneously. July 26: Patient slightly better; knee cleaner; temperature 102·4° F., pulse 108; 50 c.c. anti-*perfringens* serum. July 27: 50 c.c. anti-*perfringens* serum; improvement in general condition. July 28: 50 c.c. anti-*perfringens* serum. July 29: 50 c.c. anti-*perfringens* serum. August 1: Pulse and temperature kept up; œdema of right leg; operation, long incision; œdematous condition of leg; general improvement. September 30: Wounds healing well; re-amputation followed by very serious symptoms; pulse 160, temperature 102° F.; wound opened up. November 20: General condition excellent; right knee-joint stiff. Bacteriological report: Wound.—*Bacillus perfringens, Bacillus sporogenes, Bacillus œdematiens.* Blood.—*Bacillus perfringens, Bacillus histolyticus;* non-pathogenic anaërobe.

In association with gas gangrene streptococcal septicæmia is a very fatal complication. Although a few cases have recovered it has been usually after amputation. The action of a polyvalent antistreptococcic serum in all these cases has been disappointing. The following case is illustrative:—

Le Soldat L., aged 18, wounded on July 7, at 4 a.m., was admitted on July 10 with a gangrenous, foul smelling, penetrating shell wound of the right foot, and fracture of the os calcis and scaphoid. A large piece of shell was lying near the head of the os calcis. Operation on July 10. The projectile was removed with loose pieces of bone, and good drainage established. *Bacillus perfringens* and probably *Vibrion septique* with streptococci were reported in the wound. Two days later there was more œdema and further incisions were made. July 21: Headache; temperature 103° F. July 22: Temperature 105° F.; some bronchitic râles at both bases. July 24: Œdema over dorsum of foot; operation; some localized pus found; streptococci found in blood. July 25: 50 c.c. antistreptococcic serum, subcutaneously. July 26: Swelling of leg to knee-joint and extension of inflammation into ankle-joint; 50 c.c. antistreptococcic serum (Pasteur). Amputation of leg below knee. Examination of foot showed pus in ankle and tarsal articulations extending up tendon sheaths and much gelatinous œdema. After amputation temperature dropped and patient made a good recovery.

In this case large doses of polyvalent antistreptococcic serum were given, but with apparently little benefit.

In a certain number of cases the presence of metastatic abscesses has shown the probability that gas organisms may be circulating in the blood even when the lesion is apparently localized. These are nearly always situated at the site of some tissue injury such as a simple fracture or a subcutaneous injection. The following is an interesting example :—

Le Soldat E., aged 38, wounded on July 1, at 9 a.m., by shell, was admitted on July 3 with gangrene of the left leg as far as the knee-joint, with comminuted fracture of both bones. Crackling and œdema were present, and there was no pulse at the ankle. Both bones of the right leg were also fractured. Numerous anaërobes, including *Bacillus perfringens*, were found in discharge from the wound. July 3: Amputation through lower third of left thigh. The right leg was put on a splint and dressed with iodine. On July 8 there was extensive swelling of the right leg. The same day an incision was made and a large amount of thin serous pus let out, which contained *Bacillus perfringens* but no cocci. The case did well and finally united, though in bad position. An osteotomy of the tibia was done on November 9, and the position improved by removal of an overlapping piece of fibula.

In other cases the streptococcus localizes itself with the formation of pyæmic abscesses, while examination of the blood may give a negative result.

Le Soldat A. K., an Arab, received multiple shell wounds on July 7, at 1.30 p.m. On admission on July 8 the left leg was swollen from foot to knee, and there were numerous penetrating wounds. On July 9 several pieces of shell were removed from the foot and leg, leaving one near the head of the fifth metatarsal. An abundant *Bacillus perfringens* infection was reported. The wounds healed, but the patient could not walk, and on October 3 the piece of shell was removed from the foot under direct examination by the X-rays. October 10: The patient had a rigor; temperature, 104·6° F. The wound in the foot was sterile and looked healthy. On October 12, pain in the groin where a mass of glands could be felt. October 16: Swelling and œdema near ankle; Chopart's amputation; pus found in the calcaneo-navicular joint and a fracture of the os calcis, not previously noted, was found. Bacteriological report, streptococci and staphylococci. October 27: Pain in back; temperature, 104° F.; blood culture negative. October 29: An incision was made over the left buttock, when pus escaped from the interior of the pelvis through the great sacro-sciatic foramen. Streptococci and staphylococci were found. The case did well.

An example of the complication of a streptococcal septicæmia, not only with gas gangrene but with masked tetanus, is the following :—

Le Soldat P., aged 21, wounded by shell on September 12, with fracture of both bones of the right leg, and gas-infected wounds of right thigh and

buttock. The patient was treated in an ambulance, and put into plaster thirty-six hours before admission to Royaumont on September 17. The convoy arrived at midnight, and the patients all were very tired and collapsed, having been travelling since 6 a.m. The patient was given a subcutaneous saline and morphia. It was clear that gas gangrene was present for the tissues of the thigh were bulging over the plaster casing and the smell was foul; with considerable difficulty the plaster was removed. There was gangrene of leg and thigh, and œdema reached to the abdomen. In the early morning of September 18 a circular amputation through the upper third of the thigh was done with tension incisions through infected tissues. An enormous open wound of the right loin was cleansed. The patient stood the operation well. Bacteriological report: Mixed infection of streptococcus, *Bacillus perfringens*, and numerous other organisms. September 20: General condition had greatly improved, but the stump was swollen and very dirty; further tension incisions were made. September 23: Chains of streptococci were recovered from blood culture. September 24: The muscles of the stump were bulging, but there was no apparent gas; temperature, 103° F., pulse 114; 5 c.c. Leclainche and Vallée[1] serum applied locally to the wound. September 25: Further Leclainche and Vallée serum, 10 c.c. locally and 5 c.c. subcutaneously. September 26: Signs of pleurisy; abdominal distension with tympanites; offensive stools; 80 c.c. anti-*œdematiens* serum after further bacteriological report. September 27: 30 c.c. antistreptococcic serum; 60 c.c. anti-*œdematiens* serum. September 28: 30 c.c. antistreptococcic serum; 60 c.c. anti-*œdematiens* serum. September 29: General condition rather improved, when sudden attack took place, suggesting pulmonary embolus. The patient rallied in an extraordinary manner, and in the evening the temperature was 98° F., and pulse 88. On a report from the Pasteur Institute of the presence of tetanus, 30 c.c. antitetanic serum were given subcutaneously. September 30: Sudden pain in left eye; panophthalmitis; temperature 103° F., pulse 112; eye enucleated; contained pure culture of streptococcus; 30 c.c. antitetanic serum; 30 c.c. antistreptococcic serum. October 3: General condition improved; still rhonchi in chest and dyspnœic attacks; local condition of wounds very clean; 30 c.c. antitetanic serum. October 11: Signs of pulmonary embolus. October 13: Pericarditis. October 15: Pulse 140; pneumonia both bases. October 19: Death. Dr. Dalyell reported *Bacillus perfringens*, *Bacillus sporogenes*, *Bacillus Hibler IX*, *Bacillus œdematiens* and streptococcus, and Dr. Weinberg reported later the isolation of *Bacillus tetanus* and *Bacillus œdematiens*.

In this case the gas infection, though a most virulent one, subsided after amputation and the administration of serum. In the same way tetanus did not show itself except by slight trismus and was also controlled by serum. The streptococcus, however, in spite of amputation and the administration of two strains of serum, Pasteur, and Leclainche

[1] Rapport de E. Quénu sur la sérum polyvalent des MM. Leclainche et Vallée. *Bull. et Mém. de la Soc. de Chir. de Paris*, August 1, 1916.

and Vallée, reached the hæmic system and caused death finally by extensive implication of the lungs and pericardium.

This case was one of the most disappointing ever treated in the hospital. The assistant surgeon and sisters in charge of the patient spared themselves no pains to promote the patient's recovery, and were again and again encouraged by a temporary rally.

In another case of very extensive gas gangrene of the thigh with a mixed anaërobic infection, the patient died from a *pyocyaneus* septicæmia. The gangrenous processes had completely subsided after local excision of muscle and administration of serum.

Le Soldat C., aged 42, wounded on August 28 by shell in the right thigh, was dressed at once, and admitted to Royaumont in the afternoon of August 29. The upper two-thirds of the right thigh were much swollen, and there was some discoloration of the anterior and external surface. There was no fracture. There was a large wound over the external aspect of the right hip, below the crest of the ilium, and localized crepitation with resonance over the front of the thigh, where a foreign body was situated just external to the vessels. The patient was much exhausted. Temperature 101° F., pulse 108. Colour good. Operation at 6 p.m. The wound over the hip was enlarged, and much gaseous pus evacuated. An incision was made over the abscess, which was situated in vastus internus; gas, pus and the foreign body were evacuated. The muscle was swollen and unhealthy. Through drainage was established. The wound was washed out with hot B.P. chlorinated soda half strength. Rectal salines, strychnine, and subcutaneous salines were given with 10 c.c. anti-*perfringens* serum and 25 c.c. anti-*œdematiens*, on a report of severe anaërobic infection. August 30: Condition fairly good; temperature 101° F., pulse 118; wound dirty. August 31: Temperature subnormal, pulse 120; masses of external thigh muscles gangrenous; under anæsthesia these were cut away; 20 oz. of hot saline were given intravenously, and 25 c.c. anti-*perfringens* serum and 25 c.c. anti-*Vibrion septique* subcutaneously. In the afternoon the patient had a rigor; pulse 140, temperature 102° F.; a continuous drip was put on. September 1: Temperature subnormal; skin dark round the wound, but not crepitant; cut away freely, leaving large open wound; smell very offensive. September 2: Slight improvement; pulse 112, stronger, temperature 100° F.; 25 c.c. anti-*perfringens* serum, 25 c.c. anti-*Vibrion septique*. At this date diarrhœa began and continued without any intermission until death. On September 5 the wound was clean, but the calf became swollen and white; the foot was warm. On September 6 the patient became weak and restless. There was cough, with purulent sputum, and death took place at 10 p.m. Post-mortem: The wound was clean and the muscle tissue healthy. The femoral artery was normal, but the femoral vein was occluded by a large thrombus. There was no retro-peritoneal gaseous cellulitis. The lungs showed signs of broncho-pneumonia. The bacteriological report gave on culture

from the wound, *Bacillus perfringens*, *Bacillus sporogenes*, probably *Vibrion septique*, streptococcus, and from the blood *Bacillus pyocyaneus*.

This also was an extremely disappointing case. At one stage there seemed to be every prospect that the patient would recover when the gangrenous process was arrested.

In septicæmic cases when the streptococcus localizes itself with the formation of a fixation abscess (as for instance at the site of a subcutaneous saline injection), the process seems to be beneficial, and to increase the resisting power of the patient to the infection. The temperature usually drops after the incision of the abscess.

MORTALITY.

		Mortality	Percentage
Number of soldiers admitted ...	2,267 ...	48 ...	2
Number of cases of gas infection ...	464 ...	42 ...	9

Analysis.

(1) Gas gangrene	... ...	25
(2) Gas infection	...	
(*a*) With fracture	10	
(*b*) With brain injury ...	2	
(*c*) With abdominal wound ...	1	
(*d*) With tetanus	4 ...	17
	17 ...	42

It will be seen, therefore, that cases of anaërobic infection accounted for a large proportion of the total hospital mortality—viz., forty-two cases out of forty-eight.

TREATMENT OF CASES OF GAS INFECTION AND GAS GANGRENE.

(I) General.
(II) Surgical.
(III) Serological.

(I) General Treatment.

On admission steps are taken to make the patients as warm and comfortable as possible with the minimum of fatigue. They are usually worn out and need sleep. After being cleansed, the wound is dressed, and a bacteriological swab is taken. The patient, if the case is urgent, is then passed to the X-ray room and the necessary photographs or localizations are done. If there is no urgency he is allowed to sleep until the morning. (The majority of the cases arrive in the evening or

during the night.) If requiring immediate treatment the patient is taken to the theatre from the adjoining X-ray room and operated upon at once. If there is so much collapse as to negative operation, subcutaneous or rectal salines are given, with suitable stimulants, and the patient is left to sleep for a few hours before any operative measures are undertaken.

(II) Surgical Treatment of Wounds with Gas Infection.

A rough bacteriological diagnosis from a smear from the wound, as to the presence of many Gram-positive bacilli and streptococci, accompanies the patient to the theatre with the X-ray plates. If the wound is recent and superficial, it is excised entirely and sewn up, but we receive very few cases sufficiently early to be amenable to this form of treatment. The wounds are fully opened up by cutting away bruised and infected tissue so as to make the wound conical, and foreign bodies, including portions of clothing, are removed. In addition, in the case of a perforating wound, a piece of gauze, soaked in iodine, is drawn through the wound to cleanse its surface mechanically. Damaged skin and gangrenous tissue in the track of the projectile are cut away, large drainage tubes containing a wick of gauze soaked in salt and carbolic acid are inserted, and a special effort is made to establish through drainage by counter incisions. The opened-up wound is also packed with sterile gauze, moistened with a mixture of 5 per cent. common salt and $2\frac{1}{2}$ per cent. carbolic acid, and gauze and wool applied under the limb to soak up the copious serous discharge. Very few bandages are used, so that there is no tension, and the limb or wound can be watched.

In the first few months of 1915, the wounds were dressed with 5 per cent. salt alone, no antiseptic being used. This was not found to be entirely satisfactory; some pyogenic bacteria flourished, especially *Bacillus pyocyaneus*, and it was painful. The addition of a small percentage of carbolic acid obviated these difficulties and for eighteen months has so far proved superior to all other dressings, such as Carrel's various solutions and eusol, which have also been given a trial. It has been used successfully in many hundreds of cases and cleanses very dirty wounds in a few days. It does not, however, entirely prevent streptococcal infection, which is much more difficult to treat, and a modified form of iodine vaccine therapy[1] was tried on a number of

[1] This method differs from that described by Dr. Weinberg, *Proc. Roy. Soc. Med.*, 1916, ix (Occ. Lect.), p. 139.

Arab patients in one ward. After preliminary surgical treatment of the wounds, they were packed with iodoform gauze soaked in a weak solution of iodine: Iodine, 1 dr.; iodide of potassium, 2 dr.; aqua, 25 oz. This dressing was renewed daily. Whether the Arabs are peculiarly resistant to infection, or whether this treatment was the cause, one cannot say, but a considerable number of very badly infected cases recovered, and there was no tendency to the development of streptococcal septicæmia. Only one case of gas gangrene died, and the rest made surprisingly good recoveries. It is possible that this absorption of the products of dead microbes by the deeper tissues of the wound surface has an influence in increasing the resistance of the body. The following case is an example:—

Le Soldat A. M. received on September 12 a perforating bullet wound, which passed through the right humerus and scapula, causing extreme comminution, and a second large gangrenous wound over the left scapula. He was admitted on September 15. The wounds were exceedingly foul and evil smelling. Gas exuded from all and the surface of the large posterior wound was spongy and green. Temperature 102° F., pulse 130. The general condition of the patient was very bad, and he was considered to be on the point of death. At the operation on September 15, at 11 p.m., the shoulder wound was opened up freely, the head of the humerus and many loose pieces of bone removed. Much gangrenous muscle over the right scapula was cut away and through antero-posterior drainage established. Free skin incisions were made. The wounds were washed out with ether. The patient was very collapsed at the end, with an almost imperceptible pulse. On September 16 the infection was spreading, gas bubbled from the right scapular muscles, and crepitation was present all round the wound. At 7 p.m. (temperature 102° F., pulse 140) I again operated, removing the greater part of the scapula and scapular muscles. The muscles were gangrenous and exuded gas. The patient was much collapsed, but an intravenous saline was given before the operation, together with numerous stimulants, such as "huile camphrée," pituitrin and strychnine. To my astonishment the patient was alive in the morning, the wounds looked no worse, and there was only spread of the infection in the lower angle of the right scapular wound. *Bacillus œdematiens*, *Bacillus perfringens*, *Bacillus sporogenes*, and streptococci were reported on culture. On September 19 I cut away more gangrenous skin and muscle, and although the wounds remained extraordinarily foul there was no further spread, and no hæmic infection, although the patient was extremely yellow and anæmic and had some diarrhœa. He has made quite a good recovery, and is getting some power in the arm. After the first dressing of salt and carbolic, these wounds, together with those of the other badly infected Arabs, were dressed with 1 in 1,000 iodine on iodoform gauze.

In other badly infected cases continuous irrigation is used, with normal saline, Carrel, or salt and carbolic lotion; when healthy granulations appear iodoform gauze is applied dry. For the actual cleansing of wounds, we have found B.P. lotio sodæ chlorinatæ very useful, used with an equal quantity of hot water. It is too strong to be used as a dressing, and is a survival of the method of treatment used a good many years ago in the Royal Free Hospital for cleaning septic wounds of the hands and feet. Where there is any tendency to accumulation of pus, Saissi's T-shaped suction tubes are used with constant flow from an irrigator to form a vacuum. These have been most successful and are extremely popular, the patients themselves taking a great interest in their working. For empyema, hip- or shoulder-joint cases they are invaluable. In the hot weather wounds have been treated by exposure to the sun, with a thin dressing of gauze moist with salt and carbolic acid solution. Such cases have done very well. During the summer it has been possible to keep some of the patients entirely out of doors in beds placed round the cloisters (*see* fig. 1, p. 65). The improvement in their general condition has been most marked. For burns and where the wound involves a large superficial skin area, sterilized paraffin is used, and forms a healing painless dressing.

Treatment of Cases of Gas Infection complicated by Fracture.—Free drainage is obtained by the removal of loose fragments of bone. Where there is a linear fracture of femur or humerus with a bad infection, it has occasionally been found necessary to trephine or to remove a portion of the compact bone to provide free drainage. Often these cases with severe comminution make excellent recoveries if the infected bone is removed, the wound excised, and free incisions made down to the muscular sheaths to relieve tension. The immobilization of fractures in gas infections is often a difficulty. It is important to avoid the least pressure and tight bandages. No plaster of Paris is used. Fractures of the forearm are treated by the long, wooden, back splint suitably padded, the forearm being put up in full supination. The dressing is so arranged that the case can be watched, wool and padding being placed under the limb to absorb discharge. Most fractures of the arm are treated by Thomas's humerus splints, made with specially large rings to avoid pressure, abducted to a right angle. The case can then be dressed with a minimum of movement if the supporting flannels or zinc sheeting are well arranged. Fractures of the femur are also treated if possible with Thomas's splints, with extra large rings. Hodgen's splints have also been used for fractures of the neck of the

femur, and for cases of excision of the hip, as suitable abduction frames were delayed in transit. Wallace-Maybury splints have been found useful. For chronic bone sinuses we have recently used Morison's[1] paste of iodoform and bismuth with astonishingly good results. The cases have healed more rapidly than under any other form of treatment.

Treatment of Cases of Gas Wound Infection, complicated by Joint Injuries.—Gas infections of the shoulder have been treated in some cases by excision of the head of the humerus; occasionally drainage with fixation has been sufficient, the arm being abducted to get good range of movement should ankylosis take place. Injuries of the elbow-joint have been treated by complete or partial excision, removal of foreign bodies, and very free drainage. In a certain number of cases a considerable amount of movement has been recovered. In some the elbow has been fixed on a Jones's aluminium elbow splint with connecting half loop and suspended from a Balkan splint. The patient can then move in bed freely, and there is less tendency to œdema. Wrist-joints have been treated by partial excision, with fixation of the wrist in a position of dorsiflexion on a Jones's splint. Gas infection of the hip-joint has proved a fatal complication. Recovery took place in one instance only, where a resection was done with very free incisions. In another case it was done after conservative treatment had been adopted, and was too late to avert the rapid spread of gas gangrene. Early resection appears to give the best results, with very free posterior drainage. Infections of the knee-joint have been treated by lateral, patellar incisions, and removal of foreign bodies and loose pieces of bone. The joint is washed out and drained for a few days down to, but not into, the joint. The limb is placed on a Thomas's splint, with slight flexion of the knee-joint. In many cases good movement has been obtained. Occasionally it has been necessary to divide the patella transversely to explore the joint cavity, wiring it afterwards. Amputation has been done three times for purulent arthritis with intractable streptococcal infection, threatening septicæmia. Wounds of the ankle-joint have been treated by drainage or astragalectomy. Occasionally amputation has been required. Infections of the tarsus have been treated by drainage or partial tarsectomy. In three instances a mid-tarsal amputation was performed.

[1] "The Treatment of Infected Suppurating War Wounds," *Lancet*, August 12, 1916.

Treatment of Gas Gangrene.—When gas gangrene has supervened in the whole limb, amputation is a necessity. Except in one or two cases, where injury to the vessels was probably the primary cause of the gangrene, and the gas infection secondary, a circular amputation was performed. No attempt was made to cut flaps through infected tissues. The skin is slightly retracted upwards as the muscles are incised. My experience of flap operations has not been happy nor encouraging. In one case amputation had been done at an ambulance immediately after the infliction of the wound, in the upper third of the arm, by long lateral flaps, which on arrival were so enormously swollen that all drainage from the infected bone was impossible. The patient, in spite of repeated amputations, passed through a very critical time. In another, in amputating through the arm for gangrene of the forearm, I made slight flaps. The flaps swelled up, and in spite of repeated operations the patient died. The case was one of a virulent type, but I think the flaps were partly responsible for the fatal result, as every other case of gangrene of the forearm recovered. In operating, a tourniquet is applied for the shortest possible time, and, to save time, is usually held and not fastened. Lateral incisions are made almost invariably, so that there is no intramuscular tension in the stump, and no tendency in streptococcal cases for pus to track upwards. In the cases operated upon "*en saucisson*" this not infrequently happens. Amputation through infected tissues enables one to be more conservative, as in Case 60 (where removal of three gangrenous fingers and their metacarpals left a useful thumb and little finger). When the gangrene is localized to a group of muscles with or without bone injury, the muscular mass is excised, and the wound either dressed with salt and carbolic, or 1 in 1,000 iodine solution. In such cases in the calf, arm and forearm, some good results have been obtained. In wounds of the deepest parts of the thigh the results have been less successful. The nearer the trunk, the more dangerous is the infection, and massive gangrene involving the buttocks and pectoral regions has frequently been fatal. It cannot be too clearly realized that conservative treatment can only be adopted when it is possible to keep the patient under the closest observation. The process spreads with the most terrible rapidity, and what at night appears to be a simple localized flesh wound in the morning may have become a general infection.

AMPUTATIONS.

	Recovery	Death
Hip-joint	0 ...	2
Thigh: Upper third	1 ...	7
,, Middle third	0 ...	1
,, Lower third	8 ...	3
Leg	8 ...	0
Ankle-joint	2 ...	0
Mid-tarsal	1 ...	0
Tarso-metatarsa	3 ...	0
Arm: Shoulder-joint	0 ...	1
,, Upper third	5 ...	2
,, Middle third	4 ...	0
,, Lower third	7 ...	1
Forearm	2 ...	0
Wrist-joint	1 ...	0
Metacarpo-carpal	6 ...	0
	48	17
	Total 65	

EXCISION AND DRAINAGE.

	Recovery	Death
Excision of muscles of back	3 ...	0
,, ,, arm	6 ...	0
,, ,, forearm	3 ...	0
,, ,, leg	10 ...	0
,, ,, chest	0 ...	1
,, ,, thigh	7 ...	2
Excision of hip-joint	1 ...	0
,, ,, scapula and head of humerus	1 ...	0
,, ,, shoulder-joint	1 ...	0
Excision of muscle and drainage of penetrating pelvic wounds	1 ...	5
	33	8
	Total 41	

(*III*) *Serum Therapy.*

It is quite clear from an analysis of these cases that much remains to be done to combat the excessively high death-rate among cases of gas gangrene, both where surgery is helpless, as in cases already septicæmic, or where the wound is situated in an area not amenable to surgical treatment. The extraordinary success of antitetanic serum makes one look for help to serum specially prepared against these anaërobic gas-forming organisms.

After seeing some experimental work at the Pasteur Institute, I was anxious to try the action of serum on some of our cases. Dr. Weinberg was good enough to supply us with a quantity of special serum, which was administered in ten cases.

The difficulty that one has at once to encounter is that of knowing, in time for useful application of serum, the exact organism which is responsible for the gangrenous condition. Anaërobic organisms are by no means easy to grow, and there is still considerable confusion about their nomenclature and differentiating features. To be of use the serum must be given early. Accordingly it is necessary to give, in bad cases, a mixture of several of the most probable sera, while investigations are being carried out to determine accurately the variety of organism. My impression is that the serum has been of value as a curative measure—more particularly perhaps in cases just on the border line—to prevent the spread of the infection after amputation or excision through infected areas, and to limit the amputation, making it possible to do a transtarsal operation instead of one at the seat of election, as in the following case:—

Le Soldat V., aged 27, wounded on July 11, was dressed three hours later, and admitted on July 12, with a perforating wound of the left ankle, with fracture of the scaphoid. A very bad mixed infection was present. The wounds were enlarged, drained, and loose fragments of bone were removed. On July 17 the inner side of the foot and ankle were puffy. The scaphoid was entirely removed and better drainage established. A smear showed *Bacillus perfringens*. July 19: Temperature 100·4° F., pulse 128; foot red and swollen; mid-tarsal amputation; sinus very foul; salt and carbolic drip; no incisions into calf, which was considerably swollen as far as the knee-joint. July 20: Much more œdema of the calf, and I was tempted to amputate higher, but waited twenty-four hours to try the effect of serum, and 45 c.c. of mixed serum were injected subcutaneously. July 22: Temperature 100° F., pulse 100. July 23: Injection of 10 c.c. anti-*œdematiens* and 10 c.c. anti-*perfringens* serum. July 25: Weak iodine bath; œdema less. August 26: Healing. Bacteriological report from the wound: *Bacillus perfringens*, *Vibrion septique*, *Bacillus œdematiens*, streptococci (Dr. Weinberg).

In this case, where there was a very bad mixed infection, it was possible to amputate through infected tissues. The very considerable œdema of the leg disappeared without any incisions in forty-eight hours after two injections of serum.

In the following case also I have no doubt that the serum exerted a beneficial effect, and was instrumental in saving the life of the patient:—

L'Aspirant L., aged 27, was wounded on July 20, at 6 a.m., receiving a first dressing four hours later. There was a large antero-posterior, perforating, shell wound, which had severely comminuted the right femur in the neighbourhood of the great trochanter. On July 21 several pieces of shell were removed

and drainage established. The patient was admitted on July 23. There was a large wound on the front of the thigh, external to the vessels, and a very large, dirty, posterior wound exposing the comminuted femur. The same day the wound was dressed under an anæsthetic, and several large sequestra were removed. The wound was extremely foul, the bone visible was greyish green in colour, the surrounding skin was bruised, the muscles were gangrenous, and œdema extended on to the abdominal wall in front and over the whole of the right buttock, therefore 40 c.c. anti-*œdematiens* serum and 10 c.c. anti-*perfringens* serum were injected into the abdominal wall. On July 24 there was less œdema, though the wound was still very foul. The patient was dressed under an anæsthetic and the greater part of the great trochanter removed. This established much freer drainage. The bacteriological report on July 25 gave scanty *Bacillus perfringens* and an anaërobe not identified, probably *Bacillus œdematiens*, and *Bacillus pyocyaneus*. The blood culture was negative, although the patient was slightly jaundiced. On July 26, 40 c.c. anti-*œdematiens* serum and 40 c.c. anti-*perfringens* were given subcutaneously. Temperature 101° F., pulse 120. The enormous wound drained freely. For two or three days a dressing of salt and carbolic was used, but the wound was first washed out with ether and a solution of chlorinated soda. In about three days the dressing was changed to iodoform gauze wrung out of weak iodine (1 in 1,000), and improvement rapidly took place. A small pocket of pus running down the hamstrings was treated by a Saissi's suction tube. The wound healed steadily. The fracture was treated by a Hodgen's splint with weight extension, and united in ten weeks with about 1 in. of shortening. In this case, until the serum had been given, there appeared to be little hope of recovery.

In all ten cases only have been treated with serum. Five ended fatally, but in one of these death resulted from an aërobic septicæmia, the gangrenous process having previously yielded to treatment, and three were patients with advanced gas septicæmia who died before a correct bacteriological diagnosis could be made. In five successful cases, four were also amputated, but in addition, one had a triple hæmic, anaërobic infection, together with a gas infection of the remaining knee-joint, and in the two cases already described large œdematous areas remained after operation. It is unwise to base any opinion on a few cases, but I am convinced that the application of serum therapy will be of great value in reducing the mortality from this terrible complication of war wounds, more especially if it can be utilized prophylactically in the same way as antitetanic serum. It is essential that sufficiently powerful sera should be obtained.

In the administration of serum, anaphylaxis has been guarded against by the injection of preliminary small doses, or by local wound

application of serum, when an interval of more than ten days has elapsed since the last administration. In few cases have serious anaphylactic symptoms been noted, and in one only do I think it contributed to a fatal termination. In this case a minute dose of antistreptococcal serum was given intravenously to a patient with a severe hip-joint infection who was already *in extremis*.

In conclusion, I wish to express my grateful thanks to my assistant surgeons, Drs. Nicholson, Berry, Ross, Wilson, Courtauld, Hodson, and Martland, for their untiring assistance and co-operation; to Dr. Agnes Savill and her assistant radiologists, and to Drs. Butler, Dalyell, Oswald, and Estcourt, for their bacteriological reports. My thanks are also due to Colonel Cousserge (Médecin-chef, Gare Régulatrice de C.), for his unflagging sympathy and encouragement; to Dr. Weinberg (Institut Pasteur) for his personal help, and to Médecin-Inspecteur Général Sieur (Service de Santé) for permission to produce this paper.

In addition I am indebted to Miss Vera Collum for X-ray prints, and to Orderly Ramsay-Smith for much clerical assistance.

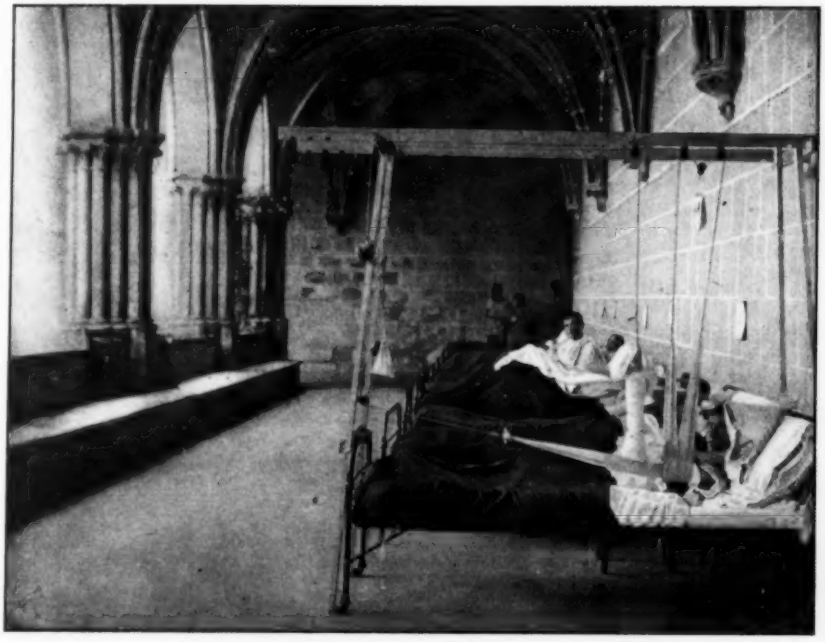

FIG. 1.

Open-air treatment of the wounded in the cloisters (Abbaye de Royaumont).

FIG. 2. (Case 4.

Gas-bubbles in and near middle metacarpo-phalangeal joint..

FIG 3. (Case 9.)

Multiple shell wounds of buttock and thigh. Excision of gangrenous muscles. Recovery.

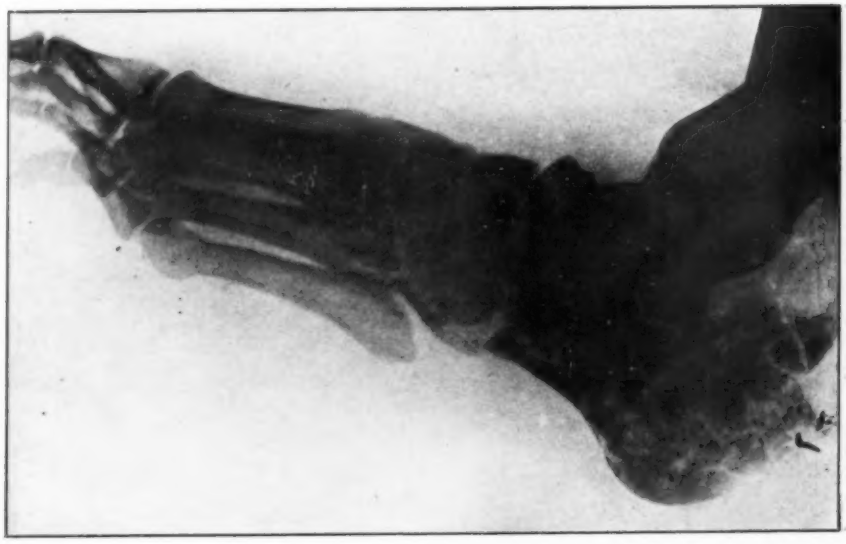

FIG. 4. (Case 21.

Fracture of os calcis, cuboid, astragalus, and three metatarsals. Gas infiltration of heel.

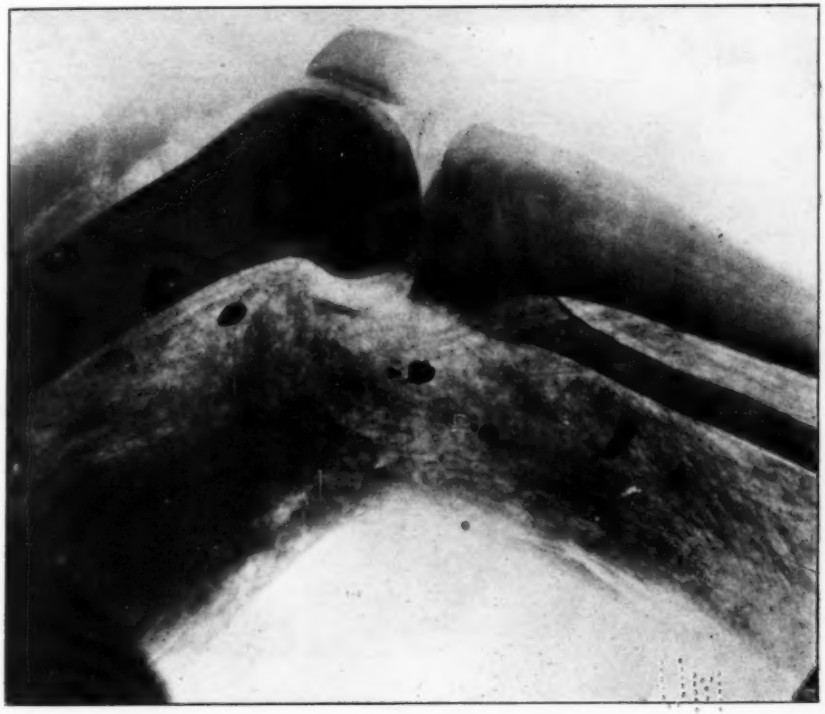

FIG. 5. (Case 27.)

Class I, classic form: Gas gangrene of knee-joint and leg. Striation and gas-bubbles with separation of muscle fibres. *Bacillus perfringens, Bacillus histolyticus, Bacillus sporogenes* present in wound, *Bacillus perfringens* in blood. Death.

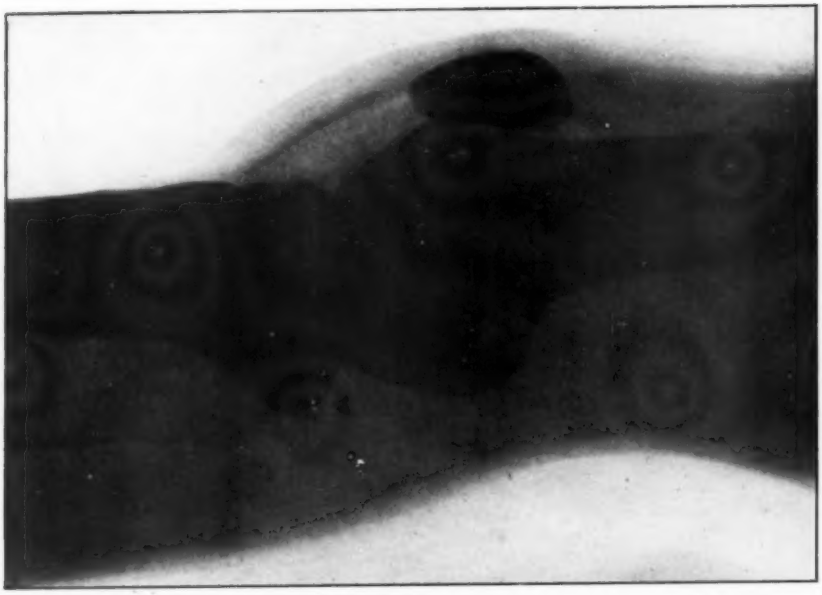

FIG. 6. (Case 31.)

Shell wound of right knee-joint. Gas in joint. Triple anaërobic blood infection. Recovery after serum treatment.

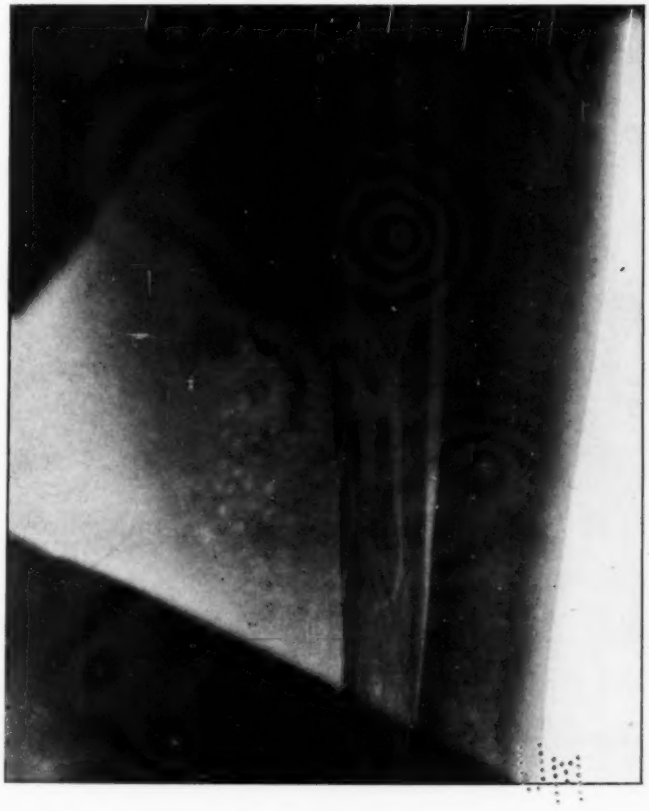

FIG. 7. (Case 41.)

Localized gas gangrene of calf with fractured fibula. Gas-bubbles. Excision of muscle and part of fibula. Recovery.

FIG. 8. (Case 52.)

Comminuted fracture of left humerus, showing gas in the neighbourhood of the fracture. Recovery without amputation.

FIG. 9. (Case 58.)

Localized gas gangrene of peroneal muscles. Superficial gas-bubbles. Recovery after excision of gangrenous muscles.

FIG. 10. (Case 97.)

Class IV. —Gas abscess in popliteal space near shell.

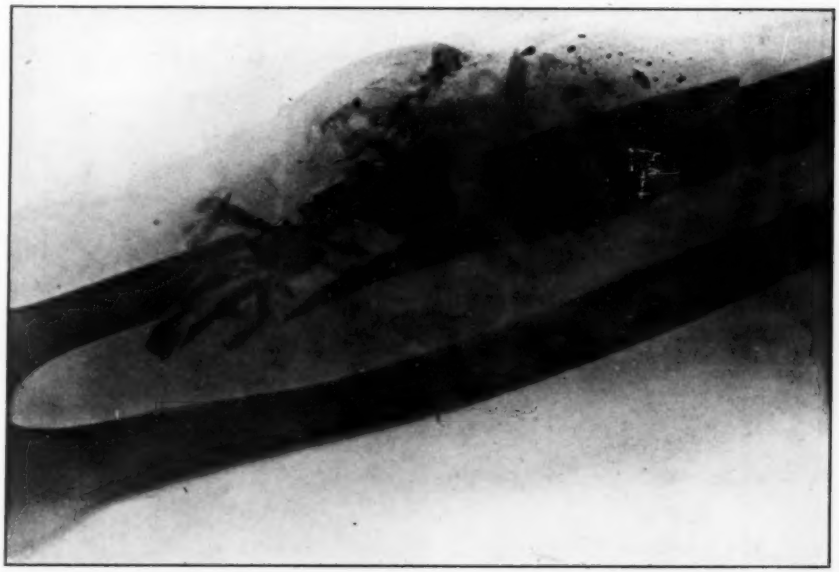

FIG. 11. (Case J.)

Class V.—Gas phlegmon. Comminuted fracture of the ulna. Recovery after drainage and incision of muscle sheaths.

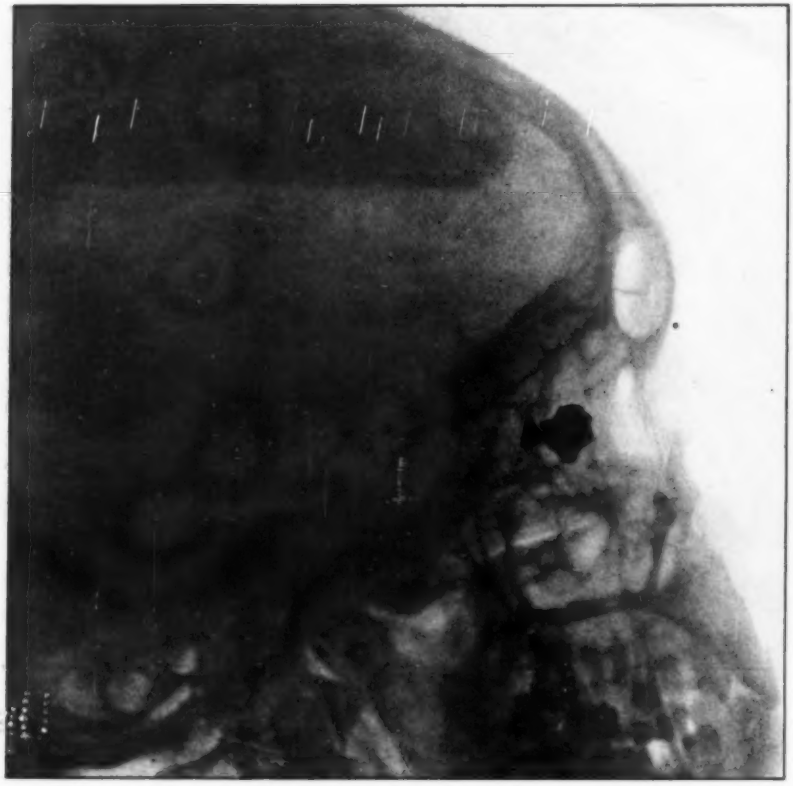

FIG. 12. (Case L.)

Class VI.—Latent infection. Shell removed from ethmoid four months after receipt of wound. *Bacillus perfringens* and streptococci present.

FIG. 13. (Case 106.)

Class VI. Delayed gas gangrene. Compound fracture of tibia. Striation. Photograph taken ten days before amputation.

An Analysis of 107 Cases of Gas Gangrene.

Abbreviations: T. = Temperature; P. = Pulse; R. = Respiration. * = Report from Dr. Weinberg (Pasteur Institute).

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
1	L., 34, French (Dr. Wilson)	July 1, 9.30 a.m., shell	July 2, 24 hours after being wounded: penetrating wound of right shoulder and arm with discoloration of skin and brawny swelling of arm; open gangrenous wound of right scapular region, thin hæmorrhagic, foul discharge and gas; severe pain right arm, lymphangitis, yellow; collapsed; T. 98° F., P. 100. July 3: Very much yellow gelatinous infiltration of cellular tissue of right shoulder and pectoral region, pus formation in scapular wound, arm more swollen and crepitant, foul dark discharge; hæmorrhagic vomiting, dyspnœa, conscious almost to end; T. 97° F., P. 130	July 2: Operation; projectile and capote removed; tension incisions. July 3: Amputation upper third right arm, incisions over deltoid and pectoralis major through deep fascia; dressing of 5 per cent. salt and 2½ per cent. carbolic; subcutaneous saline	Streptococcus, *Bacillus perfringens*	*Bacillus perfringens*	Streptococcus; taken day of admission, July 2	Fracture of right humerus, large piece of shell in arm, arm swollen	3 days; death July 4 at 6.30 a.m.; mixed toxic and classic form; infection of the deep arm muscles caused œdematous type of gangrene with later development of classic type; coincident streptococcal infection
2	C., 42, French (Dr. Nicholson)	July 1, 10 p.m., shell	July 2, 24 hours after being wounded: general condition good; T. 102·6° F., P. 102; perforating wound of left arm and forearm with dirty, ragged edges, exposing gangrenous extensor carpi ulnaris, supinator longus, extensor carpi radialis; biceps cut across; brachial artery thrombosed; hand and forearm blue and swollen; no radial pulse felt	July 2: Operation; amputation of left arm in lower third by circular method with very short flaps; dressing of salt and carbolic	*Bacillus perfringens* and other anaërobes; cocci; Gram negative sporing rods	*Bacillus perfringens*	—	(I) No fracture or foreign body; irregular tracery and swelling; gas bubbles; July 2, 1916	14 days; evacuated healing, July 14, 1916; classic form. In this case, which was of a mild type, the gangrene was primarily due to the obstruction o the blood stream
3	V., 21, French (Dr. Nicholson)	July 1, 9 a.m., shell	July 2, morning, 24 hours after being wounded: general condition good; open gangrenous wound of left thumb which was nearly torn off through metacarpal; thumb gangrenous; T. 99·2° F.; P. 104	July 2: Operation; amputation of left thumb with metacarpal; left widely open; dressing of salt and carbolic	*Bacillus perfringens*, cocci	*Bacillus perfringens*, heavy growth	—	(I) Comminuted fracture of first left metacarpal; shows fuzziness in thumb muscles; July 2, 1916	14 days; healing; evacuated July 14, 1916; classic form

4	D., 30, Senegalese (Dr. Courtauld) (*see* fig. 2, p. 66)	July 1, shell	July 2, 24 hours after being wounded: general condition good; perforating wound of left hand with fracture of head of middle metacarpal and gangrene of finger; much gas and pus present; very painful; foul smell; T., 101·4° F.; P., 112	July 2: Operation; amputation of finger with part of third metacarpal; Dressing of salt and carbolic	*Bacillus perfringens*, scanty organisms	*Bacillus perfringens*	—	(I) Comminuted fracture of middle left metacarpal into joint; gas bubbles; July 2, 1916	14 days; healing; evacuated July 14, 1916; classic form
5	M., 22, French (Dr. Ross)	June 30, 1 a.m., shell	July 2: General condition fair: T. 101° F., P. 112; perforating wound of right upper arm, extending from anterior border of biceps to middle of deltoid; very infected; brachial artery divided; pulse at wrist very weak; exposed muscles gangrenous; loss of sensation in thumb and index middle fingers. September 13: Median nerve compressed	Operation July 2: Gangrenous muscle cut away; tension incisions; brachial artery ligatured; dressing of salt and carbolic. October 23: Operation; liberation of median nerve	*Bacillus perfringens* and other sporing anaërobes	*Bacillus perfringens*	—	(I) Upper arm shows a few gas bubbles, July 2, 1916	4 months; localized classic form; in this case, although there was a bad infection and the brachial artery was ligatured, the gangrene was circumscribed
6	G., 33, French (Dr. Ross)	July 1, 10.30 a.m., shell	July 13: General condition poor, skin yellow; T. 103° F., P. 100; flaps of stump much swollen and unhealthy, skin reddened on shoulder, gelatinous infiltration. August 2: Extreme œdema over shoulder, flaps swollen and adherent; spreading streptococcal skin infection. August 9: T. 104° F.; flaps again excessively swollen; bone inflamed; offensive discharge	August 1: Incisions. August 2: Amputation of flaps and piece of humerus; superficial skin incisions through 10 per cent. iodine. August 9: Amputation circular with lateral incisions; 1 in. of humerus removed. October 9: Re-amputation by flaps	Streptococcus; *Bacillus perfringens*; few organisms. August 12: Abundant streptococci, no anaërobes	Streptococcus, *Bacillus perfringens*, *Bacillus pyocyaneus*	August 8, negative	(Before admission); fracture of both bones of forearm	4 months; œdematous type (not dressed for 12 hours after wound; amputation, July 4, at ambulance, left arm upper third; long flaps lateral; an instance of the danger of long flaps). October 30, 1916: Stump healed
7	G., 28, French (Dr. Nicholson)	July 1, shell	July 2: General condition fair; T. 102° F., P. 88; very infected perforating wound of right thigh dividing vastus internus and rectus femoris transversely; gas; muscles and part of skin discoloured and gangrenous; foul smell	July 2: Operation; bridges of skin with gangrenous muscle cut away, and wound made open; dressed salt and carbolic	*Bacillus perfringens*, pus cells, cocci	*Bacillus perfringens*	—	Shows nothing	21 days; localized classic form; healing; evacuated July 21
8	T., Senegalese (Dr. Courtauld)	July 1, shell	July 2: Condition good; T. 99° F., P. 84; perforating wound of right hand with gangrene of fifth finger and metacarpal; gas and foul smell	July 2: Amputation at base of metacarpal; dressed salt and carbolic	Streptococcus, mixed anaërobic infection	—	—	(I) Comminuted fracture fifth right metacarpal; swelling; July 2, 1916	14 days; healing; evacuated July 14; classic form

No. of case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
9	K. S., 20, Senegalese (Dr. Courtauld) (*see* fig. 3, p. 67)	July 1, shell, multiple	July 3: General condition bad; T. 97° F., P. 108; collapsed: multiple penetrating wounds of right forearm, with fracture of both bones, and forearm gangrenous; gas; multiple penetrating wounds of right buttock, with muscles in gangrenous condition; foul smell. July 14: Oscillation of T. due to severe infection of buttock	July 3: Circular amputation through lower third of right arm; dressed salt and carbolic. July 20: Three pieces of shell removed from buttock. July 27: Ditto	Streptococci, *Bacillus perfringens*, *Bacillus sporogenes*, *? Vibrion septique*	*Bacillus perfringens*, streptococci	—	(I*a*) Comminuted fracture of both bones of forearm multiple shell; (I*b*) gas infiltration, July 3, 1916. (II*a*) Multiple shell in buttock; (II*b*) Superficial gas infiltration; July 3, 1916	4 months; T. finally settled; wounds cleaned. October 30: Healed; classic form involving the whole of forearm and right buttock
10	M. K., Senegalese (Dr. Courtauld)	July 3, shell	July 4: Condition collapsed; T. 97° F., P. 120; large gangrenous wounds of left forearm; forearm swollen and crepitant to elbow-joint; gas and foul smell	July 4: Operation; circular amputation through middle of arm; dressed salt and carbolic; September 2: Stump trimmed	*Bacillus perfringens*, *Bacillus sporogenes*, cocci	*Bacillus perfringens*, *Bacillus sporogenes*	—	(I) Fracture of radius and ulna severely comminuted; extreme swelling and woolliness of muscles; July 4, 1916	4 months; October 30: Healed; classic form
11	A., 26, Portuguese (Dr. Martland)	July 4, 7.30 p.m., shell	July 8: Fair condition; T. 98·4° F., P. 72; glancing wound of left foot, with gangrenous tendons, swelling of foot and ankle; foul gaseous discharge. July 19: Foot swollen and red. August 23: Pus in scapho-cuneiform articulation; base of first metatarsal free	July 8: Drainage. July 20: Second toe with metatarsal and part of internal cuneiform removed. August 23: Drainage; dressed salt and carbolic	*Bacillus perfringens*, *Bacillus coliform* cocci	*Bacillus perfringens*. August 25: Streptococci, Staphylococci	—	Fracture of second metatarsal and internal cuneiform	4 months. October 30: Healed, union; local mixed form; removal of the second metatarsal with internal cuneiform gave good drainage
12	A. S., Senegalese (Dr. Savill)	July 20, shell	July 23, 1 o'clock: General condition bad; T. 100° F., P. 120, running; comminuted fracture of right fibula and lower jaw; right leg cold and gangrenous, distended by gas to knee-joint; thigh swollen slightly; foul smell. July 24: Stump clean, not swollen in morning; T. 100° F., P. 128; delirious; 6 p.m., suddenly unconscious, enormous swelling of stump	July 23: amputation at 2.15 p.m. by circular method, upper thigh with lateral incisions; fractured bone removed from jaw; dressed salt and carbolic	*Bacillus perfringens*, *? œdematiens*, *Vibrion septique*, 4 p.m. July 23	*Vibrion septique* in muscles at seat of amputation	*Bacillus perfringens*,* *Vibrion septique*;* taken at operation	(I) Fracture of fibula; much gas in tissues; shows streaks and lines of gas both deep and superficially in lines of muscles; July 23, 1916.	4 days; death from septicæmia July 24, at 7 p.m.; previous operation in ambulance on fibula; classic form
					Antiperfringens 12 c.c. Antiœdematiens 30 c.c. Anti-*Vibrion septique* 30 c.c. Followed by 2 pints of saline subcutaneously				

13	B., ? 21, Senegalese (Dr. Courtauld)	July 1, shell	July 3: Condition poor; T. 101·8° F., P. 112; perforating wound of right thigh, with fracture of head of femur, and perforating wound of right hand, with gangrenous muscles of buttock (entry wound); much gas, foul smell. July 10: Gangrene of right limb; restlessness; T. 103·4° F., P. 120; succumbed shortly after operation	July 3: Operation; incisions, sequestra removed; drainage; much gangrenous muscle cut away. July 10: Amputation through hip-joint after ligature of common femoral; intravenous saline	*Bacillus perfringens*, streptococci	*Bacillus perfringens*	—	(I) Comminuted fracture of neck of femur; gas infiltration; July 3, 1916	10 days; death July 10, at 6.50 p.m.; classic form of gas gangrene; delayed onset
14	G., ? Senegalese (Dr. Savill)	July 8, shell	July 11: Poor condition; T. 98° F., P. 96; perforating wound of right calf, with fracture of fibula and localized gangrene of muscles of calf, chiefly peronei; offensive gaseous discharge. July 13: Further swelling of calf; T. 101° F.; fairly clean	July 11: Leg wounds enlarged and masses of gangrenous muscle cut away; sequestrotomy. July 13: incisions; dressing salt and carbolic	Anaërobic infection	*Bacillus perfringens*	—	(I) Fracture of right fibula; swelling	103 days. October 19: Returned to Front; localized classic form; good functional result
15	D. S. Senegalese (Dr. Savill)	July 1, shell	July 3: Collapsed; T. 99° F., P. 108; perforating wound of the left elbow-joint, with much gaseous infiltration. July 5: Gas infiltration of arm; T. 103° F. July 6: T. 104° F., P. 102	July 4: Resection with removal of lower end of humerus and olecranon; drainage. July 5: Amputation lower third of arm, open. August 10: Re-amputation	*Bacillus perfringens* very abundant; no spores	*Bacillus perfringens*	—	Fracture of lower end of humerus and olecranon	4 months. October 30: Healed; classic form
16	D. D., Senegalese (Dr. Savill)	June 29, shell	July 4: Condition good; T. 99° F., P. 92; wound of middle third of left leg, with gas exuding from wound; leg gangrenous in lower part. July 18: Secondary hæmorrhage. September 13: Abscess round vessels	July 5: Amputation through upper third of left leg, left open; salt and carbolic. July 18: Re-amputation lower third thigh; flaps which were opened up. September 2: Re-amputation by flaps. September 13: Opened and drained	Severe mixed anaërobic infection	*Bacillus perfringens*, *Bacillus sporogenes*, streptococci	—	Fracture of tibia and fibula; no photograph	112 days. October 18: Evacuated, healing; leg in plaster on arrival; classic form

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness result; remarks
					Smear	Culture	Blood		
17	S. D., 23, Senegalese (Dr. Savill)	July 1, shell	July 4: T. 102° F., P. 84; fair condition; perforating wound of the left leg extending into knee-joint, with gangrenous muscles of leg; knee-joint swollen; foul gaseous discharge; severe infection of right thigh, gangrenous wound of posterior surface. August 5: Abscess formed on inner side of stump. September 13: Sinus present. September 14: T. 101·8° F.	July 5: Left leg, circular amputation through lower third of femur. August 5: Abscess opened and drained; Saissi's tube. September 13: Re-amputation by flaps	*Bacillus perfringens*, *Bacillus sporogenes*, other anaërobes, abundant streptococcal chains, numerous staphylococci	*Bacillus perfringens*, *Bacillus sporogenes*	—	(I) Fracture of tibia and fibula into knee-joint; very extensive swelling and gaseous infiltration; July 5, 1916, woolliness	4 months. October 30: Healing; classic form; infection of knee-joint
18	J.-J., 33, French (Dr. Hodson)	July 3, 9 a.m., shell	July 4: Condition poor; septicæmic appearance; yellow skin; T. 99·4° F., P. 100; multiple shell wounds of left forearm, right calf, right buttock, and gangrene of left leg; wound of buttock; foul gaseous discharge from all wounds. July 8: T. 102·8° F., P. 112. July 30: Large popliteal abscess on both sides; stump unhealthy. The condition of the patient was precarious for weeks; improved when placed out of doors	July 5: Amputation left leg by flaps; drainage; sequestrotomy of right ischial tuberosity. July 30; Popliteal abscesses opened on both sides; salt and carbolic dressing	*Bacillus perfringens*, streptococci, staphylococci	*Bacillus perfringens*, streptococci, staphylococci	October 10: Streptococcus	(I) Comminuted fracture of left tibia and fibula; scattered shell; fracture of right ischial tuberosity; shell in left forearm and pelvis; shows swelling and gaseous infiltration; July 4, 1916	4 months; healed, except for sinus in buttock. (First dressing 9 hours after wound, shell removed, leg put in plaster to mid-thigh.) Classic form; associated streptococcal septicæmia; flap operation failed (popliteal abscess)
19	D., 33, French (Dr. Berry)	July 1, bullet	July 2: Good condition; T. 98·4° F., P. 106; perforating bullet wound of left arm, with small wounds of entrance and exit; much swelling; gaseous, dark discharge. July 5: Hand painful, blue, pulseless; swelling and œdema to elbow	July 3: Sequestrotomy, drainage, arm put up in supination. July 5: Amputation middle of arm with short open flaps; dressing salt and carbolic	Streptococcus, *Bacillus perfringens*, *Bacillus sporogenes*	*Bacillus perfringens*, *Bacillus sporogenes*	—	(I) Comminuted fracture of radius and ulna into elbow-joint; swelling, tracery bubbles; July 3, 1916	24 days. July 21: Healed by first intention; evacuated July 24; (dressed 4 hours after being wounded); an early stage classic form; the extremely sudden onset suggested a vascular lesion; amputation by short open flaps gave good result

20	B., 22, French (Dr. Nicholson)	July 2, bullet	July 4: Condition fair; T. 99° F., P. 96: perforating wound of right gluteal region and sacro-iliac joint; discoloration and undermining of skin and muscle; foul muscular slough. July 6: Wound very unhealthy; symptoms of cord involvement; retention of urine; delirium; head retraction; T. 103·6° F., P. 108; distension of abdomen; skin yellow; smell of gangrene most foul; much gas exuded from wound; great distension, post mortem	July 5: Sinus between wounds laid freely open by excising gangrenous muscle; fragments of bone removed; salt and carbolic dressing	*Bacillus perfringens*, streptococcus	*Bacillus perfringens*, streptococcus	Streptococcus, July 7	Fracture of left iliac bone and sacrum through sacro-iliac joint; particles of bullet casing; X-ray failure	8 days; death on July 10 at 11.40 p.m. from septicæmia and meningitis; classic form
21	B., 21, (Dr. Berry) (*see* fig. 4, p. 68)	July 4, 9 p.m., bullet	July 6: T. 103° F., P. 120, good; comminuted fracture of right foot, involving tarsus and metatarsus; foot blue and much swollen; some blebs. August 13: Foot which had been clean became swollen and gangrenous, extremely painful; much yellow œdema and gas round heel; T. 104° F.; swollen gland in right groin. August 16: Leg swollen and red; wound of foot gangrenous. August 18: Arms stiff	July 6: Foot opened up along outer side; amputation of third toe; sequestrotomy. August 17: Amputation ankle-joint; salt drip. November 11: Re-amputation	*Bacillus perfringens*, streptococci	Streptococcus, *Bacillus coliform*, *Bacillus perfringens*, *Bacillus sporogenes*, others unidentified	—	(Ia) Fracture of os calcis, cuboid, astragalus, second, third, and fourth metatarsals; extreme comminution and gas infiltration of os calcis; bubbles; (Ib) gas infiltration and swelling of heel, July 6, 1916; (II) swelling of foot, August 11, 1916	4½ months. November 20: Healed. (First dressing 15 hours after wound.) Latent tetanus; stiffness of jaw and muscular rigidity 40 days after wound; mixed type of gangrene
					August 13: Antistreptococcic serum 10 c.c. August 15: Antitetanic serum 5 c.c. subcutaneously. August 16: Antitetanic serum 20 c.c. subcutaneously				
22	P., 37, French (Dr. Wilson)	July 9, 3 p.m., bullet	July 11, morning: T. 101·6° F., P. 108; fair condition; perforating wound of left calf, fungating; leg swollen and crepitant from ankle to knee, with mottled discoloration of upper half of calf; swollen muscle, and foul discharge exuding from exit wound. July 12: T. 98·4° F., P. 120; foul smelling pus at back of stump. July 25: Healthy granulations	July 11: Amputation below knee by circular method, through swollen skin. August 29: skin grafting by flap method	Anaërobic infection, *Bacillus perfringens*, staphylococcus, streptococcus	*Bacillus perfringens*	—	(Ia) Comminuted fracture of tibia and fibula; gas infiltration; swelling; July 11, 1916	4½ months. November 20: Healing; classic form

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
23	S. R., 24, French (Dr. Nicholson)	July 9, 2 p.m., shell	July 11: Poor condition; T. 101·6° F., P. 100; large lacerated wound over lower and outer aspect of right biceps into elbow-joint, very foul, and discharge dark; large incised wound of triceps, with swelling of arm above; tissues gangrenous, muscles discoloured on outer side; serous discharge. September 5: Wound healed, after re-amputation, by first intention	July 11: Amputation middle of arm with fascial tension incision over deltoid. September 5: Re-amputation. Salt and carbolic dressing	Streptococcus, *Bacillus perfringens*, *Bacillus sporogenes*	*Bacillus perfringens*, *Bacillus sporogenes*	—	(*Ia*) Fracture of lower end of humerus; two pieces of shell in elbow joint; shows appearance of gas bubbles. (*Ib*) Tracery and gas bubbles; July 11, 1916	83 days; healed by first intention. September 30, discharged. Œdematous form
24	G., 24, French (Dr. Wilson)	July 10 shell	July 12: Exhausted, yellow, cyanosed; T. 103° F., P. 128, R. 40; perforating wound of back through pleura and lung near lower angle of scapula; muscles gangrenous; pus exuding from wound in posterior axillary line; signs of right pneumonia; R. 40. July 15: Increasing gangrene of back; restless	July 12: Wound enlarged and loose fractured rib removed. July 15: Distress in breathing increased; dressed salt and carbolic	Cocci	*Bacillus perfringens*, *Bacillus sporogenes*	*Bacillus œdematiens* July 16	Fracture of right fifth and sixth ribs; too ill for photograph	5 days. July 15: Sudden death from septic pneumonia; gas septicæmia
25	M. b. M., 18, Arab (Dr. Nicholson)	July 6, 4 p.m., shell	July 13: T. 99° F., P. 90; lacerated wound of left forearm with torn muscles; extensor carpi ulnaris and flexor sublimis; gas and fragments of bone in wound; foul smell; lacerated wound of left hand. July 16: Gangrene of forearm; T., 103° F., P. 116	July 13: Drainage; amputation of fourth and fifth fingers. July 16: Amputation upper third of forearm; dressed salt and carbolic; September 11: Re-amputation followed by inflammatory reaction	*Bacillus perfringens* (scanty), streptococci	*Bacillus perfringens*, streptococci	—	(I) Fracture of ulna, fourth and fifth metacarpals and unciform; July 13, 1916; second plate deficient	102 days. October 16: Discharged, stump healed. (Dressed ½ hour after wound; 4 days at G.) Classic form with involvement of wrist-joint

26	G., 26, French (Dr. Ross)	July 9, bullet	July 11: T. 103° F., P. 106, good; hand swollen; pus in wrist joint. July 17: Secondary hæmorrhage from hand. July 22: Extreme swelling of forearm; much serosity; carpus infiltrated with foul pus	July 14: Wounds enlarged; drained; sequestra removed. July 17: Amputation three outer fingers. July 22: Amputation upper third of forearm; dressed salt and carbolic	Slight anaërobic infection, *Bacillus perfringens*	*Bacillus perfringens*	Negative	(I) Fracture of second, third, and fourth metacarpals, os magnum, scaphoid; extreme swelling of hand; July 12, 1916	81 days. September 26: Stump healthy. September 28: Healing; œdematous form
27	B., 20, French (Dr. Wilson) (*see* fig. 5, p. 69)	July 16, noon, bullet	July 17, midnight: Condition poor; T. 102° F., P. 128; yellow, restless; penetrating, foul smelling wound of right leg and left foot; right limb swollen and crepitant to Poupart; foot cold; bullæ distended with dark coloured fluid in leg; above knee swollen and crepitant, but not discoloured; no swelling of abdomen; gangrenous wound of left foot; rectal saline, &c., 3 a.m.; thigh more swollen. July 18: No further swelling in stump	July 18: Amputation 3 a.m., through upper third of thigh; gas escaped with violence; tourniquet slipped, but artery controlled by finger; no hæmorrhage; pulseless at end of operation; revived temporarily, but gangrene extended to left foot	*Bacillus perfringens*, *Bacillus sporogenes*, and other forms not identified; severe anaërobic infection	*Bacillus perfringens*,* *Bacillus histolyticus*,* *Bacillus sporogenes*	July 18, 3 a.m., *Bacillus perfringens**	(I) Multiple shell and much gas in thigh and leg; gas in articulation; striation fine and coarse; gas bubbles; much separation of muscle fibres; July 17, immediately after amputation	3 days. July 19, 6 a.m., death from anaërobic septicæmia
28	D., 34, French (Dr. Nicholson)	July 16, 6 p.m., shell	July 18: T. 102·8° F., P. 102; perforating wounds of left calf (with hæmorrhagic discharge, bulging of muscle), passing between bones; soleus gangrenous; much swelling of calf; gas; foul smell	July 18: 5.20 p.m., gangrenous muscle (soleus) excised; salt and carbolic dressing	*Bacillus perfringens*, cocci, bacilli in pus cells	*Bacillus perfringens*	—	Fracture of fibula; some swelling of leg only	79 days. October 3: Discharged, wounds healing. (First dressing ½ hour after wound; two dressings before admission.) Localized classic form
29	R., 24, French (Dr. Wilson)	July 10, 2 a.m., shell	July 10, 10 p.m.: T. 99·2° F., P. 132; collapsed, yellow, frequent vomiting; foul smelling wound of right forearm, with two large openings; extensive gangrene of both flexor and extensor muscles; muscles swollen and protruding; rectal salines given. July 11, early morning: Pulse improving and more regular before operation. July 27: Some suppuration in stump. September 6: Streptococcal infection after re-amputation	July 10: Amputation lower third of humerus. September 6: re-amputation with drainage; T. 102·6° F., P. 128; salt and carbolic dressing	*Bacillus perfringens*, *Bacillus sporogenes*, ? *Bacillus œdematiens*	*Bacillus perfringens*	*Bacillus œdematiens**	None made; fracture of radius and ulna	91 days. October 30: Healing; mixed œdematous and classic form; infection of blood evidently transient as T. dropped after amputation

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
30	D., 20, French (Dr. Wilson)	July 20, shell	July 21: T. 98·4° F., P. 72; lacerated and gangrenous wound of left foot, with loss of second, third and fourth toes, and laceration of big and little toes; foot discoloured and swollen; foul discharge; some gas	July 21: Amputation of big toe through metatarsal and little toe through metatarso-phalangeal joint; salt and carbolic dressing	Scanty *Bacillus perfringens*, streptococcus	*Bacillus perfringens*, streptococcus	Negative	(I) Destruction of toes of left foot; July 21, 1916	66 days. October 20: Healing; classic form (localized)
31	G., 21, French (Dr. Nicholson) (*see* fig. 6, p. 70)	July 19, midnight, shell	July 21, 6 p.m.: Bad condition, yellow; T. 101·2° F., P. 130; perforating wound of left leg with discoloration of skin of leg, cold; engorged veins and swelling of thigh; wounds of right knee-joint. July 25: Swelling of right knee-joint; T. 103° F., P. 102; masses of gelatinous material evacuated; much yellow œdema of thigh along internal saphenous vein; muscles gelatinous. August 1: Much œdema of right leg. September 14: T. 103·4° F., P. 112. October 11: Re-amputation followed by very severe collapse; P. 160, intermittent, T. 101° F.; wound opened up; systolic murmur; no dilatation. October 14: Great improvement. October 20: No murmur; patient can move right leg	July 21: 9 p.m., amputation left thigh; fractured bone removed with *capote* from head of tibia. July 25: Knee joint drained. August 1: Incisions into leg. August 7: Put in Thomas's splint. September 15: Abscess in popliteal space opened. October 11: Re-amputation	*Bacillus perfringens*, *Bacillus sporogenes*, ? *Bacillus œdematiens*	*Bacillus perfringens*, *Bacillus histolyticus* *Bacillus sporogenes* *Bacillus œdematiens*	*Bacillus perfringens*,* *Bacillus histolyticus*, putrid anaërobe* not completely studied. July 23: *Bacillus perfringens*. July 24: Negative. July 25: *Bacillus perfringens*, and other anaërobes. July 28: Negative. July 29: *Bacillus perfringens*, *Bacillus histolyticus*. August 2: Negative	(Ia) Fracture of left tibia and fibula by shell, with gas round shell, deep-seated; (Ib) gas tracery more superficial; July 21, 1916. (II) Fracture of right tibia by shell. (III) Shows gas bubbles in right knee-joint; July 21, 1916	November 16: Healed. (First dressing 12 hours after wound. July 20: Left leg and right knee put in plaster.) Mixed classic and œdematous form; gas septicæmia followed by recovery

July 22: 25 c.c. antiperfringens, 25 c.c. antiœdematiens, subcutaneously. July 23: 32 c.c. antiperfringens, 75 c.c. anti-*Vibrion septique* and antiœdematiens subcutaneously. July 24: 40 c.c. antiperfringens, 70 c.c. œdematiens, subcutaneously; 30 c.c. into wound. July 25: 110 c.c. antiperfringens. July 28: 50 c.c. antiperfringens. July 29: 50 c.c. antiperfringens.

32	C., 21, French (Dr. Ross)	July 15, shell	July 16: Condition good; T. 98.4° F., P. 72; infected wound on plantar aspect of right foot, inner border; foot swollen and purplish-red; vessels divided; gas. July 22: Inner part of foot gangrenous; foul discharge; œdema extends above ankle	July 16: Wound opened up; internal cuneiform and shell removed. July 22: Amputation; remains of internal cuneiform, great toe, and base of second metatarsal débris and *capote* removed	*Bacillus perfringens*, *Bacillus streptococcus*, very abundant	*Bacillus perfringens*, Streptococcus	—	(Ia) Fracture of right internal cuneiform by shell; fracture of bases of second and third metatarsals; (Ib) Some gas tracery in sole; July 16, 1916	80 days. October 3: discharged; healing wound, good movement; localized œdematous form
33	L., 18, Arab (Dr. Nicholson)	July 7, shell	July 10: Condition good; T. 98·4° F., P. 72; dirty foul smelling wound on dorsum of right foot; foot much swollen; gas. July 12: T. 101·6° F., P. 96; much swelling. July 19: T. 105·4° F.; headache, bronchial râles. July 24: Streptococci in blood. July 26: Swelling of leg to knee, with gelatinous infiltration; purulent and gaseous infiltration of foot	July 11: Fractured bone removed with sequestra; drained. July 24: Head of os calcis and piece of scaphoid removed. July 26: Amputation of leg, upper third; drip. November 9: Re-amputation, transcondylar	*Bacillus perfringens*, *Bacillus sporogenes*	Streptococcus, *Bacillus perfringens*, *Bacillus sporogenes* *Bacillus histolyticus*	Streptococcus, July 24	(I) Fracture of scaphoid and head of os calcis by shell; July 10, 1916. (IIa) Shows disorganization of calcaneo-cuboid articulation. (IIb) Gas infiltration round os calcis; July 23, 1916.	4 months. November 22: healing. (First dressing ½ hour after wound.) Septicæmia; much pus found in ankle joint and extending up tendons of leg; œdematous form
					July 25: 50 c.c. antistreptococcic serum subcutaneously. July 26: 50 c.c. antistreptococcic serum subcutaneously				
34	P., 27. French (Dr. Nicholson)	August 3, 3 p.m., gun carriage over foot	August 4, midnight: T. 98° F., P. 72, good; gangrene of left great and second toes, with swelling on dorsum of foot; third and fourth toes swollen; foul smell, gas. August 11: antitetanic serum rash	August 5: Amputation of second, third, and fourth, toes at metatarso-phalangeal joints, and of great toe through proximal phalanx; drainage; salt and carbolic dressing; drip	*Bacillus perfringens*, Staphylococcus	*Bacillus perfringens*, *Bacillus coliformis*, Staphylococcus	—	Fracture of first and second toes (phalanges)	41 days; discharged September 13, healing wound. (First dressing ½ hour after wound.) Local œdematous form

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result remarks
					Smear	Culture	Blood		
35	R., French (Dr. Nicholson)	August 25, 3 p.m., shell	August 29: T. 101·6° F., P. 80, exhausted; penetrating wound of right hand, with dirty wound over thenar eminence; hand swollen and discoloured; smell penetratingly offensive, gaseous pus; multiple wounds of scalp, ear, left leg, foot, and thorax. September 1: Forearm swollen, wound foul; T. 102·2° F.	August 29: Right hand removed, leaving fifth finger; trapezium, trapezoid, os magnum removed. September 1; Amputation wrist-joint, long incisions; drip	*Bacillus perfringens*, *Vibrion septique*, *Bacillus sporogenes*, *Bacillus coliform*, Staphylococcus	*Bacillus perfringens*, *Vibrion septique*, Staphylococcus, *Bacillus coliformis*	—	(I) Large piece of shell in hand; shows fracture of carpus, and fourth metacarpal, swelling and tracery; August 29	3 months; October 30: All wounds almost healed. (First dressing 1 hour after being wounded.) Classic form; extremely severe infection; interval between wound and operation, 4 days
					August 25 — Antiperfringens serum 25 c.c. Anticœdematiens serum 25 c.c.				
36	E. A., 36, Arab (Dr. Hodson)	July 6, shell	July 8: Penetrating wound of left elbow-joint, swollen to middle of arm, great pain. July 11: Tissues of forearm œdematous; gas and foul smell	July 9: Joint opened, fragments of ulna removed; July 10: Incisions. July 11: Amputation lower third arm. September 8: Re-amputation	*Bacillus perfringens*, *Bacillus sporogenes*	*Bacillus perfringens*	July 17, negative	Fracture of ulna; two fractured bones in elbow-joint	86 days; healing, good stump: œdematous type
37	F., 21, French (Dr. Nicholson)	July 8, shell bullet	July 10: Fair condition; T. 100° F., P. 104, yellow; wound of right thigh and left foot, extensive, foul, involving almost whole right thigh, and exposing lacerated hamstrings and vastus externus; no crepitation nor discoloration; left foot, wound of sole across metatarsal bones. July 11: Wound dirty, no swelling or discoloration. July 12, 10 a.m.: No swelling, but uneasiness in leg; later, severe pain; 2 p.m., blue discoloration above and below wound; swelling of thigh. July 14: Died; purple discoloration of skin at site of subcutaneous injections	July 10; Infected muscles cut away; shell removed; salt and carbolic dressing. July 12: Amputation upper third of thigh, circular, with lateral incisions; gas exuding	*Bacillus perfringens*, other anaërobes	*Bacillus perfringens*, *Vibrion septique*	July 12, *Vibrion septique*,* *Bacillus œdematiens**	(I) Right thigh with shell; some gas infiltration tracery, July 10; (II) left foot showing metallic particles in wound of sole, and fracture of all metatarsals	6 days; death July 14. (First dressing ½ hour after wound.) A very acute case; pain probably due to the sudden escape of gas from deep muscles through burst sheaths; mixed form; gas septicæmia

38	F., 21, French (Dr. Ross)	July 8, 11 a.m., bullet	July 10: Condition poor; T. 104° F., P. 124; perforating calf wound of left leg with commencing gangrene of muscle, swollen, tender; offensive smell; pus. July 14: Leg swollen above knee, gangrenous to knee, crepitation and skin discoloration in popliteal space; foot cold. July 19: Pyæmic abscesses of hand, sacrum; very mild symptoms of tetanus; irritability of temper	July 10: Gangrenous muscle cut away. July 14: Amputation thigh in lower third	Streptococci abundant, *Bacillus perfringens* scanty	*Bacillus œdematiens,* Bacillus histolyticus,* Bacillus tetanus**	Streptococcus, July 14	(I) Fracture of tibia; swelling only; July 10, 1916	13 days; death, July 25. (First dressing July 9, 5 a.m.) Death from streptococcal septicæmia; classic form
					Pyæmic abscesses contain pure streptococcus; July 17: Antistreptococcic serum 25 c.c. July 19: Antistreptococcic serum 40 c.c. July 21: Antistreptococcic serum 60 c.c. July 23: Antistreptococcic serum 50 c.c.				
39	C., 20, French (Dr. Berry)	July 8, bullet	July 11: Good condition; T. 99° F., P. 84; perforating wound of right buttock, with comminution of neck of femur; wounds minute; some pain on micturition. July 15: Some swelling; gas in sinus. July 16: Gas and œdema; T. 102·6° F., P. 104. July 17: After amputation progressive multiplication of gas in stump; T. 99·8° F., P. 128; skin yellow	July 13: Wounds enlarged, sequestrotomy. July 15: Excision of head of femur. July 16: Amputation at hip-joint; rallied; P. 128. July 17: Died	Pus cells; slight *Bacillus perfringens*; no cocci or other organisms	*Bacillus perfringens*	—	(I) Comminuted fracture of neck of femur and pelvis; July 12, 1916	9 days; death July 17. (First dressing 3 hours after wound. Bullet removed from posterior wound before admission), mixed form, beginning as deeply seated œdema
40	P., 32, French (Dr. Nicholson)	July 1, 6 a.m., shell	July 12: T. 100° F., P. 104; poor condition; perforating wound of middle third of right thigh, drained, with foul blue gaseous pus; penetrating wound of thorax (air and pus bubbling out); left eye, perforating wound with perforation of cornea and protrusion of iris; penetrating wound of first interphalangeal joint of left middle finger	July 13: Thigh wound cleaned; left eye enucleated; chest wound drained; left middle finger amputated. July 14: Thigh swollen. July 15: Foot blue, cold; mid-day, amputation site of fracture; patient died two hours later; did not rally	*Bacillus perfringens*, mixed anaërobic infection, streptococci	*Bacillus perfringens*	? *Bacillus œdematiens* taken post mortem July 16	(Ia) Fracture of middle third of right femur; (Ib) some tracery near tube; July 13, 1916	15 days; death, July 15 (four pieces of shell removed at V——; thigh in plaster): œdematous form; septicæmia

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
41	L., 23, 23, French (Dr. Nicholson) (*see* fig. 7, p. 71)	July 16, 9 a.m., shell	July 18, 1 a.m.: T. 100·6° F., P. 106; condition good; perforating wound of left leg, calf swollen, gas infected, muscles bulging out of wound; foul smell. August 28: Acute osteomyelitis of fibula. August 30: More swelling of calf	July 18: Wounds opened up, soleus cut out and peronei; sequestrotomy; salt and carbolic. August 28: Abscess in fibula evacuated, drained. August 30; Further incisions. October 6: Abscess opened near upper extremity of fibula	Streptococci, *Bacillus perfringens*, *Bacillus sporogenes*	Streptococcus, *Bacillus perfringens*	Negative	(I) Comminuted fracture of fibula, left middle third; localized gas in neighbourhood of fractured fibula, with (II) gas bubbles in muscles and in subcutaneous tissue; July 18, 1916	6 months; healed; fibula re-formed. (First dressing ¼ hour after wound.) Classic form (local)
					August 28: Antistreptococcic serum 60 c.c.				
42	C., 42, French (Dr. Ross)	July 16, 6 p.m., shell	July 18: T. 99° F., P. 90; large open wound over back of right arm and upper end of ulna; very dirty, with gangrenous muscle; *capote* embedded in wound; radial pulse felt; foul discharge. July 18 (night): complete gangrene of forearm	July 18 (early morning): Muscles cut away, sequestrotomy. July 18: Amputation lower third of humerus; circular, no flaps. September 13: Scar excised	*Bacillus perfringens*, severe mixed anaërobic infection	*Bacillus perfringens*, *Bacillus sporogenes*	—	(I) Ulna fractured; much swelling in upper part of forearm, with gas infiltration and bubbles in ragged muscle; July 17, 1916	91 days; discharged October 5; good stump; ulnar nerve divided; classic form
43	D., French (Dr. Wilson)	July 20, shell bullet	July 20, midnight: T. 102° F., P. 120; good condition; perforating wound of left calf and superficial wound of right leg; left calf swollen to ankle but not red nor crepitant; on outer side of middle of calf, muscles gangrenous, swollen and protruding from wound: August 12: Severe urticarial rash	July 21: Gangrenous soleus, and outer head of gastrocnemius cut away	*Bacillus perfringens*, severe mixed infection	*Bacillus perfringens*, *Bacillus sporogenes*	Negative	(I) Swelling of calf; one or two gas bubbles; slight tracery; July 21, 1916	40 days; healing; evacuated August 29; œdematous form (local)

44	V., 44, French (Dr. Rose)	July 13, 12.30 p.m., shell	July 14: Good condition; T. 98·4° F., P. 72; gaping wound over left tendo-Achillis, gangrenous, shell size of walnut protruding. July 21: Periostitis of tibia. July 26: Foot swollen, œdema to calf, and discoloration of skin; much serous infiltration; gas, foul odour; infection of ankle-joint. August 20: Slow healing	July 14: Projectile removed. July 21: Wound opened up; gangrenous skin cut away, piece of stocking and sequestrum removed. July 26: Amputation upper third of leg, circular. September 30: re-amputation	*Bacillus perfringens*, streptococcus	*Bacillus perfringens*, *Bacillus sporogenes*	—	Shell near external malleolus; fracture of internal malleolus	4 months; November 16: stump healed; œdematous type rather latent; ankle joint opened up by fracture
45	W., 21, French (Dr. Nicholson)	August 25, bullet	August 29: T. 99·4° F., P. 100; pale, collapsed; perforating wound of left sacroiliac region from A. S. spine to buttock. August 30: No gas seen. September 4: Œdema of buttock; foul smelling discharge; T. 103° F., P. 124; much yellow œdema and staining of tissues. September 5: Opisthotonus, restless, twitching; P. 150, delirium, T. 103° F.; vomiting	August 30: Both wounds opened; sequestrotomy; drainage September 4: Long incisions over trochanter, bone removed; packed salt and carbolic	Scanty *Bacillus perfringens*, staphylococci, coliform organisms;	*Bacillus perfringens*, scanty streptococci	*Vibrion septique* (from blood post mortem, September 5)	(I) Comminuted fracture of ilium above acetabulum; August 30, 1916	11 days; death September 5, at 5.30 a.m. (First dressing ½ hour after wound.) Post mortem: gas in iliopsoas; muscles gangrenous and like lung; I. V. C. filled gas; acetabulum fractured into hip-joint. Mixed form, probably also tetanus
					September 4: Antiperfringens serum, 75 c.c.; Anticœdematiens serum, 75 c.c.; Anti-*Vibrion septique* serum, 75 c.c.				
					September 5: Antitetanic serum, 20 c.c., 30 c.c.				
46	Le C., 21, French (Dr. Berry)	? July 9, shell	July 10, midnight: Collapsed, delirious; T. 98° F., P. 150; skin yellow; numerous wounds of right leg; leg swollen, skin discoloured, muscle dark red in colour; foul gaseous discharge from wound; five large pieces of shell present	July 11, 1 a.m.: Amputation of thigh, lower third	*Bacillus perfringens*, mixed anaërobes	*Bacillus perfringens*, *Bacillus sporogenes*	No examination of blood	Multiple shell in leg, fracture of fibula; marked streaking and tracery; July 10, 1916	? 24 hours; history uncertain; death immediately, July 11; patient extremely collapsed on admission; toxæmic form of gangrene, probably septicæmia

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
47	V., 27, Corsican (Dr. Berry)	July 11, shell	July 12: Condition good; T. 98° F., P. 128; perforating wound of left ankle, not much swelling; foot gradually became puffy and some foul gas exuded. July 19: Inner side of ankle puffy; foot reddened, sinus foul, tarsal joints affected; T. 100·4° F., P. 128. July 20: Œdema of leg to knee. July 25: Improvement	July 12: Wounds cleaned and incised, through drainage. July 17: Scaphoid removed. July 19: Amputation middle tarsal joint	*Bacillus perfringens*, ? *Bacillus œdematiens*	*Vibrion septique*,* *Bacillus perfringens*,* *Bacillus œdematiens*,* Streptococcus*	—	(Ia) Fracture of left scaphoid, swelling and tracery; (Ib) shows outline of foot with tracery; July 12.	58 days. September 7: discharged; healing. No incisions made for œdema of calf and leg, which cleared up on serum treatment; œdematous form
					July 20: Antiperfringens serum, 45 c.c.; Anticœdematiens serum, 45 c.c.				
					July 23: Antiperfringens serum, 10 c.c.; Anticœdematiens serum, 10 c.c.				
48	B., 21, French (Dr. Ross)	July 1, 8 p.m., grenade	July 3: T. 99° F., P. 138; collapsed; multiple wounds of right arm, leg, thigh, and left leg; very dirty, infected and gangrenous wound of right thigh, much muscle infected; foul gaseous discharge; condition of patient very bad owing to multiplicity of wounds	July 5: Operation, gangrenous muscle cut away. July 20: Cleansing of wounds, projectiles removed, &c. August 29: Second toe and metatarsal amputated	*Bacillus perfringens*, streptococcus	*Bacillus perfringens*, streptococcus	—	Fracture of right ulna; fracture of right fourth and fifth phalanges and os calcis; third right metacarpal; multiple projectiles	4½ months. November 20: Healing; localized classic form
49	G., 23, French (Dr. Ross)	July 2, 5 p.m., shell	July 4: Perforating wound of right forearm, very tense and swollen; gas in deep tissues; foul discharge; skin gangrenous; gas in infected perforating wound of left side of thorax	July 5: Gangrenous skin and muscle cut away from forearm; dressed with salt and carbolic	*Bacillus perfringens* and other anaërobes	*Bacillus perfringens*	—	—	3 months. September 13: Healing. October 15: Nearly healed; limitation of arm movements owing to muscle destruction; fractured bone removed before admission; œdematous form (localized)

50	M. b. H., Arab (Dr. Ross)	July 7, 3 p.m., shell	July 8: T. 101° F., P. 120; gangrenous perforating wound of left thigh, with wound of exit on inner side; offensive gaseous discharge; hamstrings divided	July 8: Wound opened up and gangrenous muscle cut away; sciatic nerve exposed	*Bacillus perfringens*, ? other anaërobes; cocci	*Bacillus perfringens*	—	Negative	19 days. July 26: Discharged, nearly healed; classic form (localized)
51	B., 26, French (Dr. Ross)	July 7, 11.30 p.m. shell	July 8: T. 102° F., P. 124; lemon yellow; ? blood infection; perforating wound of left foot with much swelling; discoloration of skin, and large gangrenous patch over heel; offensive discharge; blebs over foot and ankle; gas; simple fracture of right os calcis. July 19: Foot completely gangrenous	July 8: Gangrenous heel cut away; sequestrotomy. July 19: Amputation seat of election, no flaps. July 24: Incisions of right foot	*Bacillus perfringens*, *Bacillus sporogenes*	*Bacillus perfringens*, *Bacillus sporogenes*	Negative, July 23	(I) Comminuted fracture of left os calcis and astragalus, with gas round metallic particles. (II), simple fracture of right os calcis; July 8, 1916	62 days. September 8: Discharged, healing; classic form
52	C., 34, French (Dr. Wilson) (*see* fig. 8, p. 72)	August 24, 6 p.m., bullet	August 29: Good condition, arm swollen, reddened in upper half, no crepitation; gangrenous wound in upper third of arm on anterior surface; muscles protruding	Operation August 29; drainage, sequestrotomy. August 31: Arm swollen; biceps cut away; arm put again on Thomas's splint	Streptococcus, *Bacillus perfringens*, Staphylococcus	*Bacillus perfringens*, streptococci, *Bacillus coliformis*	—	(I) Comminuted fracture of left humerus; fuzziness; swelling very excessive in subcutaneous tissues; August 29. (II) Shows callus formation; October 12, 1916	November 3: Healing. Gas infection localized; œdematous
53	El A., Arab (Dr. Nicholson)	July 6, 6 p.m., bullet	July 8: T. 102° F., P. 112; exhausted: left leg swollen from knee to toes, small wound on inner side of left calf; big toe red and swollen; gas; extremely foul smell	July 8: Wounds connected by deep incisions through muscles, *capote* and clot (foul) evacuated; gastrocnemius and soleus cut away; eusol	Intracellular, *Bacillus perfringens*, *Bacillus sporogenes*	*Bacillus perfringens*, *Bacillus sporogenes*	—	(I) No fracture; tracery; bubbles; ? streaking; July 8, 1916	54 days. August 9: wound slowly closing in. Evacuated August 29, not healed. Local œdematous type

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
54	C., 42, French (Dr. Wilson)	August 28, 5 a.m., shell	August 29: T. 101° F., P. 108; exhausted; colour good; penetrating wound over external aspect right hip below crest of ilium; discoloration of anterior external surface of right thigh; localized crepitation over fractured bone on anterior internal surface; extremely offensive gaseous discharge. August 30: Muscles completely gangrenous on anterior external surface; T. 97° F., P. 120. August 31: Rigor. September 2: Gangrenous skin round wound excised; diarrhœa began and continued without any amelioration; T. 100° F., P. 112. September 5: Wound clean; no spread of gangrene; calf swollen and white; foot not cold. September 8: Weak and restless, cough	August 29: Wound over hip enlarged; fractured bone with gas and pus evacuated from vastus internus. August 30: Muscle cut away	Severe anaërobic infection	*Bacillus perfringens*, *Bacillus sporogenes*, streptococcus	*Bacillus pyocyaneus*, September 9	(I) Thigh; shell with gas abscess and tracery; no fracture; August 29, 1916	11 days. Death, September 8. 11 p.m., incisions. Post mortem: Wound clean; femoral artery normal; femoral vein occluded by large thrombus; recent pleural adhesions and congestion; Classic form (localized)
					August 29: Antiperfringens serum, 10 c.c.; Anticœdematiens serum, 25 c.c.				
					August 30: Antiperfringens serum, 25 c.c.; Anti-*Vibrion septique* serum, 25 c.c.				
					September 2: Antiperfringens serum, 25 c.c.; Anti-*Vibrion septique* serum, 25 c.c.				
55	D., 30, French (Dr. Ross)	August 1, 3 a.m., shell	August 5: Good condition; T. 100·8° F., P. 100; perforating wound of lower end of forearm, very dirty, yellow serosity. August 6: Looking healthy. August 7: Swelling of hand, rapidly extending in afternoon to arm. August 10: Œdema; 3 p.m., very dirty and swollen. August 16: Arm doughy; 5 p.m., swelling and serosity along course of artery, with much yellow œdema	August 5: Wound cleaned; sequestrotomy. August 7: Amputation upper third forearm; incisions. August 10: 3 p.m.: Amputation lower third right arm. August 16, 5 p.m.: Amputation 2 in. higher; bone marrow curetted. November 11: Reamputation	*Bacillus perfringens* and other anaërobes	*Bacillus perfringens*, *Bacillus Hibler IX*, streptococcus	Negative, August 7	Comminuted fracture of ulna and radius; much swelling, August 5, 1916.	2½ months; healed November 20; muscle excised before admission; typical case of œdematous or toxic gangrene
					August 5 and 7: Anticœdematiens serum 25 c.c. August 10 and 13: Anticœdematiens serum 40 c.c. August 16: Anticœdematiens serum 40 c.c., and Antistreptococcic serum 30 c.c.				
						Bacillus œdematiens,* spore-bearing* mobile aërobe			

56	G., 25, French (Dr. Wilson)	July 9, 2 p.m., shell	July 10: T. 101° F., P. 88; lacerated; perforating wound of left forearm, with gangrene affecting flexor and extensor muscles; muscles swollen and protruding from wound; deep gas; amputation considered	July 10, evening: As general condition good, arm not amputated but deep gangrenous muscles of forearm excised, and long incisions made through deep fascia to relieve tension; salt and carbolic dressing	*Bacillus perfringens*, *Bacillus sporogenes*	*Bacillus perfringens*, *Bacillus sporogenes*	—	(I) Fracture of left radius, upper third; gas bubbles in centre of swollen muscle; July 10, 1916	91 days; healed; movements of fingers and wrist improving. October 8: Evacuated; median nerve affected; œdematous form (local); conservative treatment successful in this case where interval only 28 hours
57	M., 21, French (Dr. Nicholson)	July 1, bullet	July 2: T. 101·8° F., P. 100; gangrene of forearm (right); no pulse, skin black, forearm cold; arteries torn across. July 8: Not draining well, swollen to axilla down to muscle; redness and œdema of arm; thick pus tracking under biceps. July 9: Flaps œdematous. July 14: T. 102·4° F., P. 106. July 18: Swollen to axilla, vomiting	July 2: Amputation arm, lower third, with *slight skin flaps*. July 8: Flap opened; tension incisions. July 14: Flaps cut off. July 16: Further incisions	*Bacillus perfringens* and other anaërobic forms, pus, sporing anaërobes	*Bacillus perfringens*, *Bacillus sporogenes*, ? *Bacillus histolyticus*	—	Fracture of both radius and ulna; no plate taken	19 days; death July 19; œdematous form; flap operation.
58	F., 29, French (Dr. Ross) (*see* fig. 9, p. 73)	July 2, 2.30 p.m. shell	July 3: Gas; offensive smell; T. 102° F.; perforating wound of left calf, involving peroneal muscles, which are gangrenous. July 28: Swelling in peroneal muscles	July 3: Disintegrated peroneal muscles cutaway. July 28; Some swelling of muscles, more cut away	Intense anaërobic infection, *Bacillus perfringens*, other anaërobic forms	*Bacillus perfringens*, *Bacillus sporogenes*,	—	(I) Much swelling, tracery, bubbles, of calf, July 3, 1916	78 days; discharged September 17 nearly healed, no complications; œdematous form (localized); deep gas
59	P., 32, French (Dr. Berry)	July 1, shell	July 3: Condition poor, yellow; T. 98° F., P. 120; multiple infected wounds of left thigh, one full of gaseous pus leading down to fracture of femur. July 4: Thigh swollen. July 5: Thigh enormously swollen and crepitant, extending to abdomen; much offensive discharge; P. 160; T. 103° F.; much gas. July 6: Very yellow, stump swollen and dirty. October 30: Condition gradually improved	July 3: Abscess of femur drained; Shell removed from right shoulder. July 5: Amputation upper third thigh; skin incisions in buttock opening up sinus; intravenous saline. July 6: Incisions to relieve tension; salt and carbolic dressings; Carrel drip. July 25: Shell removed from right knee-joint. October 4: Re-amputation; shell removed	July 3, *Bacillus perfringens*, numerous other anaërobes July 21; shell negative	*Bacillus perfringens* *Nil* on smear or culture; October 6, shell, showed large Gram positive bacillus and streptococcus, staphylococcus	July 25, negative	Fracture middle femur; shell in left thigh and right thigh and knee, and right shoulder-joint; defective plate	4 months. October 30: Healed; had every appearance of septicæmia; this was the only case of amputation in the upper third of the femur for gas gangrene which survived; classic form

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of place	Duration of illness; result; remarks
					Smear	Culture	Blood		
60	C., Senegalese (Dr. Courtauld)	July 1, shell	July 2: T. 102° F., P. 104; condition fair; wound of left hand with three fingers almost torn off and gangrenous to wrist; foul dark discharge. October 30: Can oppose thumb and little finger	July 2: Three affected fingers amputated with metacarpals widely open. August 15: Sequestrotomy	*Bacillus perfringens*, mixed infection, many Gram positive anaërobes thick and thin	*Bacillus perfringens*, *Bacillus sporogenes* September 15, *Bacillus perfringens*, streptococci	—	(I) Swelling of thumb muscles; destruction of middle three fingers and metacarpals, August 5, (II) after treatment shows restoration of opposition; shape restored	4 months; healing. October 30: Can pick up with thumb and little finger. November 5: Healing; local classic form
61	G., 30, French (Dr. Wilson)	July 1, shell	July 2: T. 98° F., P. 82; good condition; large open wound at insertion of left deltoid; gangrenous muscle; foul smell; gas	July 7: Gangrenous muscle cut away; Thomas's splint	Mixed anaërobic infection, numerous cocci	*Bacillus perfringens*	—	(I*a*) Fracture of humerus, oblique, no displacement; metallic debris; (I*b*) wide sinus leading to fracture with marked gas tracery and bubbles. August 1	66 days; healing September 24; classic form (localized)
62	E., 38, French (Dr. Nicholson)	July 1, 9 p.m., shell falling on both legs	July 3: Fair condition; T. 96° F., P. 126; large lacerated foul wound near left knee-joint, very offensive; gangrenous muscle crepitant, no pulse. July 8: Discoloration of right leg; thin pus tracking about (*Bacillus perfringens* in pus). July 16: Abscess opened. September 15: Fracture united, bad position. November 16: Position good	July 3: Amputation thigh, left lower third, through healthy tissues, slight flaps. July 8: Incisions. July 16: Abcess incised. October 11: Re-amputation left thigh. November 9: Osteotomy, right tibia and fibula	Severe mixed infection staphylococci	Streptococci, *Bacillus perfringens*, *Bacillus sporogenes*, ? *Bacillus histolyticus*, *Bacillus perfringens* in pus of right leg, July 8	—	Fracture of left tibia and fibula; fracture of right tibia and fibula.	5 months. September 12: Fracture united. October 30: Right leg healing; stump healed; classic form; an instance of tissue injury as determining the localization of gangrene

63	B., 24, French (Dr. Martland)	August 27, shell	August 30: T. 98·6° F., P. 100; pallid; penetrating wound of left thigh on outer side, very dirty, no swelling; no gas seen at operation; hæmorrhagic discharge from muscle. August 30: Profuse hæmorrhagic discharge from muscle. August 31: Much swelling of thigh; bronzing of skin to knee; red infiltration of muscle; pus. September 2: Muscles crepitant, bulging, gangrenous; rigors, pulse bad, collapse	August 30, 1.5 p.m.: Tensor fasciæ femoris opened, projectile removed, through drainage. August 31: Part of hamstrings cut away. September 2: Gangrenous hamstrings cut away	*Bacillus perfringens*	*Bacillus sporogenes*, *Bacillus perfringens*,	No examination	No fracture; shell in left thigh	8 days; death September 4, 4 a.m. (August 28: Operation before admission; looked for shell but did not find); classic form; serum given very late
					September 2	Antiperfringens serum, 25 c.c. Antiœdematiens serum, 25 c.c.			
					September 3	Antiperfringens serum, 85 c.c. Antiœdematiens serum, 25 c.c.			
64	C., 27, French (Dr. Ross)	July 2, 2 p.m., grenade	July 3: T. 103° F., P. 104; fair. Multiple penetrating and perforating gaseous wounds of right leg below knee; leg swollen, dark blue, œdematous, crepitant; foot cold; very offensive smell	July 4: Amputation below knee-joint, circular, with oval flaps; salt and carbolic dressing	Cocci	*Bacillus perfringens*, and others	—	(I) Fracture of tibia and numerous pieces of shell in leg, July 3, 1916	58 days; evacuated August 30; wound satisfactory; classic form
65	B., 23, French (Dr. Nicholson)	July 1, shell	July 4: T. 100° F., P. 98; lacerated foul smelling wounds exposing gangrenous biceps of left thigh; external popliteal paralysis	July 5: Excision of gangrenous muscle; salt and carbolic dressing. November 4: Scar cut out, sciatic nerve isolated, bulbous portion excised, resutured, replaced in muscular sheath. November 23: Tibialis anticus and peroneus longus shortened	Severe anaërobic infection	*Bacillus perfringens*	—	No photograph	October 30; Sinus healed; classic form localized
66	F., 38, French (Dr. Ross)	July 1, bullet	July 6: T. 100° F., P. 96; lemon yellow; dry tongue; penetrating wound of right elbow-joint, arm dark red, swollen, mottled, crepitant; hand cold, no sensation; multiple wounds round elbow-joint	July 6: Amputation right upper arm, circular, open; salt and carbolic dressing	Severe mixed anaërobic infection, streptococci	*Bacillus perfringens*	—	Comminuted fracture of humerus, radius, and ulna	34 days; August 8: healing. (First dressing 5 p.m. same day. Operation July 3; arm in plaster from wrist to shoulder; window at elbow-joint); classic form

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
67	B., 36, French (Dr. Ross)	July 1, 5 p.m., bullet	July 3: T. 97·6° F., P. 50; dull and comatose; gaping wound behind the right mastoid and one to the left of the sixth dorsal vertebra; bullet beneath skin; gangrenous muscle; foul smell; gas; lethargic; reflexes normal; no incontinence	July 5: Gangrenous muscle cut away, *capote* and bullet removed. July 21: Superficial abscess opened; salt and carbolic dressing	*Bacillus perfringens*, and sporing organisms, staphylococci	*Bacillus perfringens*	—	Fracture of fifth and sixth dorsal vertebræ, sixth spinous process	101 days; August 8: Healing. October 9: Healed; slight limitation of movement of neck; classic form (localized)
68	El H., 20, Arab (Dr. Nicholson)	July 9, shell	July 10: T. 103° F., P. 116; penetrating wound of left forearm, ulnar artery divided near to olecranon, elbow swollen, very painful; large amount of red muscle degeneration; gas. July 12: Forearm cold and gangrenous	July 11: Wound of forearm opened up and shell removed; ulnar artery tied. July 12: Amputation middle third humerus, circular, open. September 11: Re-amputation	Cocci	*Bacillus perfringens*	—	(I) No fracture; shows shell and gas in neighbourhood, July 11, 1916	82 days; healing, September 29; classic form; influence of vascular obstruction in determining gangrene
69	G., French (Dr. Wilson)	July 8, 9 p.m., shell	July 11: T. 98° F., P. 96; offensive penetrating wound of right arm and thorax, with fracture of humerus and penetrating wound of left thigh; redness and swelling of shoulder; pectoralis major divided and gangrenous. July 13: Œdema of chest; much gelatinous infiltration of arm, whole shoulder much swollen; lemon yellow; T. 98° F., P. 104; arm swollen; redness and bogginess spreading to middle of sternum	July 11: Cleaned, put up on Thomas's splint; shell extracted from thigh. July 13: Gangrenous wound of chest laid open more freely; amputation of arm at level of fracture (artery occluded by blood-clot)	*Bacillus perfringens*	*Bacillus perfringens*	—	(I*a*) Comminuted fracture of humerus by shell; gas can be seen to extend on to chest wall. July 11, 1916. (I*b*) Shows commencing tracery round fragments of shell. July 11, 1916	6 days; death, July 14, 5 a.m.; died with appearance of septicæmia, sudden collapse; œdematous form; tissues gangrenous at site of subcutaneous injection
70	D., 34, French (Dr. Nicholson)	July 7, 2 p.m., shell	July 11, 5.30 p.m.: T. 99·8° F., P. 94; perforating shell wound of right calf, infecting peroneal muscles; very dirty wound; gas gangrene of peronei and tibial posticus; bronzing of skin to popliteal space	July 11: Gangrenous muscles excised and wide incisions made	Streptococcus, *Bacillus perfringens*, mixed anaërobes	*Bacillus perfringens*	—	—	54 days; September 3: Healing; can flex and extend knee-joint. (First dressing ½ hour after wound.) Classic form (localized)

71	G., 26, French (Dr. Martland)	September 13, 5 p.m., shell	September 15: T. 98·6° F., P. 100; posterior external wound of left arm, with gangrene of triceps and external part of biceps; no crepitation; serosity; very offensive	September 15: Excision of gangrenous muscle, triceps and biceps; incisions; salt and carbolic dressing. November 1: Skin grafting	*Bacillus perfringens* and spore-forming anaërobes; *Bacillus pyocyaneus*, streptococcus, staphylococcus	*Bacillus perfringens*, streptococcus	—	(I) Left shoulder and arm swelling; fuzzy appearance; September 15, 1916	2 months; November 20: Healed; œdematous form
72	P., 31, French (Dr. Wilson)	September 11, 11 a.m., shell	September 15, 6 p.m.: T. 102° F., P. 100; very yellow, exhausted; penetrating wound of right thigh in front of femur; thigh swollen, tender, swelling over middle of Poupart's ligament; drainage tube between two wounds, inner to outer surface; foul pus and gas; swelling of adductor magnus and vastus internus; femoral artery felt in wound. September 16: Patient very drowsy. September 17: Hæmorrhage femoral artery, 3.30 p.m.; much pus tracking up along great vessels towards abdomen; very collapsed after hæmorrhage	September 15: Inner wound opened and abscess found in adductor magnus; hæmorrhage in Hunter's canal; muscle cut away. September 17: Amputation upper third	*Bacillus perfringens*, ? streptococci	*Bacillus perfringens*, *Bacillus sporogenes*, *Bacillus œdematiens*	Sterile, September 17	(I) Shows abscess or gas in adductors, September 15, 1916	6 days; death, September 17, 10 p.m. (September 11, 9 p.m.: Fractured bone removed by small incision before admission.) Œdematous form, with gas in deep muscles; death from secondary hæmorrhage + toxæmia
73	C. M. ben A. Arab (Dr. Hodson)	September 12, shell	September 15: Yellow, toxæmic; T. 102° F., P. 104; collapse; large dirty wound of right leg; skin crepitant and brawny over leg; offensive discharge. September 17: Pocket of pus in soleus muscle; gas bubbles. September 20: Stump unhealthy. September 22: Gas spreading under hamstrings. September 23: Much yellow serosity, muscles hæmorrhagic; gradually failed	September 15; Amputation of right leg, upper third; drip. September 18: Re-amputation lower third femur. September 22: large pieces of hamstrings cut away	*Bacillus perfringens*, abundant aërobic spore-forming organisms	*Bacillus perfringens*, *Bacillus sporogenes*	—	(I*a*) Comminuted fracture of fibula; shell; (I*b*) gas in deep muscles; (I*c*) Extensive superficial gas infiltration. September 15, 1916	13 days; death, September 25, 6.45 a.m.; very severe infection; intractable to repeated amputations; mixed form

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
74	A. M. Arab (Dr. Hodson)	September 12, noon, bullet	September 15: T. 102° F., P. 112; very bad condition, collapse; large open wounds over both scapulæ, gangrenous, spongy, exuding gas, with fractures; most offensive odour, greenish grey; wound over anterior surface of shoulder-joint, foul smelling, and exuding bubbles of gas, edges undermined; crepitation. September 16: Infection spreading in posterior wound of right shoulder; tissues purple and spongy with gas; T. 102° F., P. 130. September 17: T. 99° F., P. 120	September 15: Wounds of right shoulder thrown freely open, head of humerus removed and many sequestra, gangrenous muscle cut away; free drainage: washed with ether, iodine, salt and carbolic dressing. September 16: Body of scapula and much of subscapularis removed, some hæmorrhage; iodine dressing; saline intravenously; muscle cut away. October 11: Sequestrotomy	Cocci, severe mixed infection	*Bacillus perfringens*, *Bacillus sporogenes*, *Bacillus œdematiens*, October 24, *Bacillus sporogenes*, *Bacillus perfringens*, *Bacillus pyocyaneus*	Sterile, July 16	(I) Comminution of right scapula, head of humerus and acromio-clavicular dislocation; extensive superficial gas tracery extending on to chest wall; great swelling; September 15, 1916	2 months; November 3: Healing; limitation of movement of shoulder; extremely severe local infection; classic form; 1 in 1,000 iodine dressings gave good results
75	P., 33, French (Dr. Ross)	September 9, 6 a.m., shell	September 16, 1 a.m.: Good condition; T. 102·8° F., P. 106; penetrating wound of lower third right thigh just above joint; wound on plantar aspect of left foot, fourth and fifth toes and muscles gangrenous; surface dirty; leg swollen, foot cold, skin of foot discoloured; crepitation; knee-joint wound very foul smelling. September 17: Signs of general infection; T. 103° F., P. 126; tongue red and dry; yellow	September 16: Shell removed right thigh; amputation lower third left thigh by circular amputation, lateral incisions; dressed salt and carbolic. November 11: Re-amputation by flaps	*Bacillus perfringens*, streptococci	*Bacillus perfringens*, streptococci	Sterile, September 18	(I) Fracture of fourth and fifth metatarsals; shell in knee-joint; cumulus cloud like gas formation; September 16, 1916	2 months; November 20: Healing; classic form
76	M., 32 32, French (Dr. Nicholson)	September 14, 1 p.m., shell	September 15, midnight: T. 99·2° F., P. 80; large lacerated foul smelling wound of left triceps, with gangrene of muscle; *capote* embedded; no swelling of arm or gangrene of skin; gas. September 16: Very dirty, swelling of elbow and edges of wound. September 21: Healing	September 16, 3 a.m.: Whole wound and skin edge excised; 1 in 1,000 iodine; salt and carbolic	Streptococcus, *Bacillus perfringens*	Streptococcus, *Bacillus perfringens*	—	Swelling only	2 months; November 20: Healed. Limited œdematous form

77	C., 28, French (Dr. Nicholson)	September 10, 2 p.m., shell, stones	September 15, midnight: T. 101·4° F., P. 88; good condition, smell extremely foul; wound of right shoulder and left leg; fœtid abscess over middle third of right fibula, tracking among gangrenous peroneal muscles; œdema of foot, reddish pus, gas; septic condition of right shoulder with large piece of shell in axilla; T. fell after operation and rapid improvement took place	September 16, 4.30 a.m.: Right leg wound laid freely open, peritoneal muscles excised freely, four stones removed (shell, right shoulder); salt and carbolic; saline drip	*Bacillus perfringens* and two other sporing anaërobes, streptococcus	*Bacillus perfringens*, *Bacillus sporogenes*, *Bacillus Hibler IX*	—	(II) Shows fracture of humerus and shell; (I*a*) stones in leg; (I*b*) striation in muscle	2 months; healing (shell localized at Amiens); limited mixed form; smell extremely foul
78	S., 38, French (Dr. Nicholson)	September 13, 3 p.m., shell	September 15, 10 a.m.: T. 101·6° F., P. 86; fair condition; lacerated wound of dorsum of left hand with swelling over third and fourth metacarpals; other superficial wounds; foul smell from wound, muscles of dorsum gangrenous.	September 15, 2.15 p.m.: Through drainage. September 16: Amputation fourth and fifth fingers through metacarpals; tension incisions in arm	*Bacillus perfringens*, numerous cocci and other anaërobes	*Bacillus perfringens*	—	Fracture of fourth and fifth metacarpals	2 months; nearly healed. Local œdematous form
79	J., 20, Belgian (Dr. Nicholson)	September 13, midnight, shell	September 16, 10.45 p.m.: T. 102·8° F., P. 86; yellow appearance; dirty foul smelling wound in right triceps and much tense swelling of muscles and subcutaneous tissues round head of humerus; redness and œdema of skin, gas near shell, œdema over shoulder. September 17: Stump clean. September 18: Gas coming from deep tissues; flaps œdematous; 7.30 p.m., poor condition. September 19: Perspiring; spreading gangrene; restless; conscious; blue; cold	September 16: Circular amputaton of right arm, incisions, September 18: Amputation at shoulder-joint, all gangrenous muscle cut away, much gas	*Bacillus perfringens*, *Bacillus sporogenes*, *? Vibrion septique*, cocci, aërobic spores	*Bacillus perfringens*, *Bacillus sporogenes*, *Bacillus œdematiens*	*Bacillus sporogenes* and unidentified bacillus	(I) Shell in right arm, gas in muscles; striation; small gas bubbles round shell, September 16, 1916	6 days; death, September 19, at 8 p.m. (September 14: Dressed A—; shell localized); Toxic and œdematous type; serum given when patient already septicæmic
				September 18: Anticœdematiens serum 50 c.c. September 19: Anticœdematiens serum, 60 and 50 c.c.					
80	K. D., Arab (Dr. Courtauld)	September 10, shell	September 15: Collapsed; T. 99·4° F., P. 80, cold; dirty perforating wound of right buttock posterior to great trochanter, already drained; sloughing, gangrenous, friable muscle; sanguineous pus; offensive smell; gas	September 15: Operation; glutei cut transversely; removal of masses of gangrenous muscle; salt and carbolic	Staphylococci, *Bacillus perfringens* and mixed anaërobes	*Bacillus perfringens*, *Bacillus sporogenes*	Sterile	No fracture; no foreign body	2 months; November 3: Healing, no complications; localized classic form

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
81	H., 26, French (Dr. Wilson)	September 13, 2 p.m., shell	September 17: T. 99·8° F., P. 84; penetrating shell wound left upper adductor region passing behind artery, with very foul discharge; no swelling nor redness; wound on inner side extending to perinæum; gas and pus round shell; muscle gangrenous on surface, but no deep infiltration	September 17: Shell removed; much infected; drainage; track leads to small trochanter; external circumflex tied; very freely laid open on each side by large incisions, and muscle cut away in track of projectile	Mixed anaërobic infection, streptococci	*Bacillus perfringens*, *Bacillus sporogenes*, aërobic spore-bearers	Sterile	Shell; fissure in femur	2 months; healing; November 3: no complications; (September 16: Operation at H.E.; drained). Localized classic form
82	D., Arab (Dr. Hodson)	September 12, shell	September 18: T. 101° F., P. 124; yellow, collapsed; perforating wound of right thigh, very dirty and foul smelling; much œdema of surrounding muscles, extending up to Poupart's ligament; gas; clean wound left thigh; right wound did not involve femoral artery. September 19: Diminished œdema; no gas seen; pneumonia left base; R. 44, T. 103·4° F., P. 130. September 29: T. normal, general improvement	September 18; Operation; wound opened and much purplish gangrenous muscle cut away; salt and carbolic dressing	Streptococcus, *Bacillus perfringens*	Streptococcus, *Bacillus perfringens*	—	(I) Gas infiltration right thigh, September 18, 1916	2 months; November 3: Wound of left thigh healed; right thigh healing; no complications. Localized œdematous form
83	D., 30, French (Dr. Martland)	September 15, 1 a.m., shell	September 16, 11 p.m.: T. 104° F., P. 120; good; dirty perforating wound of left forearm, with swelling of hand; much serosity and red infiltration; foul smell. September 18: Muscle protruding, forearm very swollen; gas exuding from muscle, serosity spreading above elbow; crepitation round wound	September 17: Sequestrotomy; muscle cut away; drainage. September 18: Amputation circular, lower third left arm; salt and carbolic dressing	*Bacillus perfringens*, very numerous diplococci, streptococci	*Bacillus perfringens*	Negative, September 18	(I) Comminuted fracture left ulna (butterfly); arm swollen, September 17, 1916	January 1, 1917: Healed; classic form
84	I., 36, French (Dr. Berry)	September 14, 2 p.m., shell	September 16: T. 100·4° F., P. 100; flesh wound on inner side of left forearm, very foul; tendons exposed, muscles grey and bulging, no crepitation, extreme œdema, redness of skin. September 22, 7 p.m.: Hæmorrhage ulnar artery	September 17: Wound laid widely open, gangrenous muscles excised; September 22; ulnar artery tied; salt and carbolic	Streptococcus *Bacillus perfringens*	Streptococcus, *Bacillus perfringens*	—	Negative	2 months; wound healing. November 3: Delayed response, ulnar nerve; early œdematous form

85	J., 23, French (Dr. Wilson)	September 14, 4.30 p.m., shell	September 17, 1 p.m.: T. 100·4° F., P. 90; fairly good condition; perforating wound 6 in. long on inner side of upper third of left thigh; skin gangrenous over sinus; localized crepitant swelling above wound; rest of thigh not swollen, slightly offensive purulent discharge. September 18: Surface rather cleaner. September 20: Swelling of muscles in wound; pain in leg, sleepless, no general swelling and no spread up abdomen; T. 101° F., P. 120; yellow, exhausted. September 21: Gas bubbling out from muscle which felt like lung tissue, very offensive; artery normal; serosity not marked; rather whiter than usual, pinker near gangrene; thigh not much swollen	September 17: Operation; gangrenous skin excised, parts of adductor brevis and magnus found gangrenous and cut away; *capote* removed; sartorius and adductor longus cut away; washed out with chlor. soda, packed with salt and carbolic. September 21: Amputation upper third of thigh	*Bacillus perfringens*, short streptococcal chains	*Bacillus perfringens*, *Bacillus sporogenes*, *Bacillus œdematiens* (muscle)	No blood cultures taken	No fracture; (I) print shows extensive gas bubbles and tracery of thigh; localized gas abscess seen in deeper tissues; September 17, 1916	7 days; death, September 21, 1 p.m. Œdematous and classic form of gangrene; long interval (3 days) occurred between wound and operation. With the onset of pain and rise of T. it is probable that gas in deeper muscles burst through sheaths
86	P., 21, French (Dr. Martland)	September 12, 7 p.m., shell	September 17, midnight: Collapsed, in great pain; T. 99·2° F., P. 98; multiple perforating wounds of right leg and thigh; wounds exuding foul pus; enormous wound in right buttock, very foul; right foot and leg tense and discoloured; much serosity right thigh, no crepitation. September 20: General condition better; stump dirty, muscles bulging; buttock wound also dirty; twitching of face. September 25: No apparent gas; T. 103° F., P. 114. September 30: Panophthalmitis (streptococcal). October 3: Embolus. October 15: Pneumonia	September 18, 11.30 a.m.: Amputation through upper third of thigh; loin wound dressed; intravenous saline. September 23: Incisions. September 30: Enucleation of eye	Mixed anaërobic infection, streptococcus	*Bacillus perfringens*, *Bacillus sporogenes*, *Bacillus œdematiens** *Bacillus tetanus*,* *Bacillus Hibler IX*	September 19, negative, September 23, streptococcus	Comminuted fracture of right tibia and fibula; no photograph	37 days; death, October 19, 1.5 p.m. (Leg put up in plaster of Paris 36 hours before admission); œdematous form of gas gangrene, which yielded to serum treatment, though the patient died of streptococcal septicæmia
					September 26: *Bacillus œdematiens* isolated. September 24: 5 c.c. Leclainche and Vallée serum locally. September 25: 10 c.c. Leclainche and Vallée serum locally. September 26: 20 c.c. Leclainche and Vallée serum locally, and 80 c.c. anticœdematiens serum subcutaneously. September 27: 70 c.c. anti-œdematiens serum, 30 c.c. anti-streptococcic serum subcutaneously; Leclainche and Vallée serum locally				

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
87	M. ben A., Arab (Dr. Hodson)	September 12, noon, shell	September 17, midnight: T. 99·4° F., P. 86; poor condition; large foul smelling perforating wounds of left upper arm with gangrene of torn ends of triceps and deltoid; ends of bone exposed; no crepitation; dirty wounds of right hand and wrist, and left hand and wrist; gangrenous right index finger; large superficial wound of left thigh	September 18: Operation; amputation left arm at seat of fracture; gangrenous muscle cut away; marrow scraped; incisions into œdematous skin of shoulder; salt and carbolic dressing; iodine; amputation of finger	Mixed anaërobic infection, streptococci, staphylococci, coliform organisms	*Bacillus perfringens*, *Bacillus sporogenes*, *Bacillus œdematiens*, *Bacillus pyocyaneus*, *Bacillus coliform* B, streptococcus	—	Not photographed on admission; comminuted fracture left humerus surgical neck	2 months; healing. November 3: Stump granulating; œdematous form; on admission arm in plaster of Paris
88	D., 28, French (Dr. Martland)	September 15, 3 p.m., shell	September 18: Good condition; T. 99·2° F., P. 84; large penetrating wound of left shoulder and back; swelling and discoloration over lower half of left scapula and below; dirty pus, gangrenous muscle, evil smelling discharge. October 20: T. fluctuating	September 18: Wound opened up completely by dividing transversely some of trapezius and latissimus dorsi; projectile removed and *capote*; salt and carbolic dressing. September 27: Iodine. October 20: Infraspinatus incised	*Bacillus perfringens*, cocci	*Bacillus perfringens*	—	Shell, fracture of seventh left rib	2 months; November 20: Healing. (First dressing 3 hours after wound. September 16: Operation at A——; shell not found.) Œdematous type; an early stage of localized gangrene; persistent streptococcal infection
89	M., 21, French (Dr. Nicholson)	September 14, 4 p.m., shell	September 17, 8 p.m.: T. 98·6° F., P. 92; exhausted, collapsed; dirty, lacerated, gangrenous wound over left deltoid and biceps; foul smelling multiple wounds of right arm and left thigh; swelling of arm; *capote* in wounds. September 18: T. 102·8° F., P. 116	September 18: Operation, at 4 p.m.; excision of gangrenous muscle; drainage; fractured bone extracted; salt and carbolic dressing	*Bacillus perfringens*, *Bacillus sporogenes*, aërobic spore forms, staphylococcus	*Bacillus perfringens*, *Bacillus sporogenes*, streptococcus	—	(I) No fracture; two projectiles posterior and external to humerus; swelling only of muscles; September 18, 1916	November 3: Healing; some difficulty in flexion; localized classic form

90	D., 30, French (Dr. Wilson)	September 7 noon, losing much blood, shell	September 18, 6 a.m.: T. 99° F., P. 74; anæmic, excitable, very ill; large open wound of right tibia upper third; no swelling of thigh or leg; large, dirty wound over left internal malleolus; foot swollen; tibial wound communicates with *knee-joint*; no gas, foul smell; wound of left foot into ankle-joint. September 19: Foot wound dirty. September 21: Right stump very dirty, adherent clot, not much swelling	September 18, 10 a.m.: Operation; foot drained; fractured bone removed, amputation lower third right thigh. September 21: Amputation left foot, midtarsal. September 23: Amputation left foot, ankle-joint, external malleolus removed; pus in ankle-joint	Mixed anaërobic infection, *Bacillus perfringens*, staphylococcus, *Bacillus coliform*	*Bacillus perfringens*, streptococcus	September 21, negative	(I) Fracture and loss of substance below head of right tibia; gas infiltration round fracture, adventitious markings in addition. (II*a*) Fracture of scaphoid by shell involving astragalo-scaphoid articulation and fracture of internal malleolus. (II*b*) Gas infiltration round ankle; September 18, 1916	Healing; evacuated November 20; requires re-amputation. Œdematous form
					September 19: 20 c.c. antistreptococcic serum subcutaneously. September 20: 40 c.c. antistreptococcic serum subcutaneously.				
91	B., 22, French (Dr. Martland)	September 16, 11 a.m., shell	September 18: Collapsed condition; T. 100° F., P. 100; dirty perforating wound of right leg; leg tense and swollen; aneurysm of post-tibial artery, gangrene of inner head of gastrocnemius and soleus; reddish, offensive discharge; discoloured from knee to ankle, not crepitant	September 18; Shell removed; ligature of right posterior tibial (ulcerated); excision of gangrenous soleus and inner head of gastrocnemius; dressed iodine 1 in 1,000	Staphylococci, *Bacillus perfringens*	*Bacillus perfringens*	—	No fracture; small fractured bone in calf	2 months; November 10: Healed; some weakness of leg owing to loss of muscle (first dressing 1 hour after wound. September 17: Incisions). Classic form, limited to calf muscles
92	P., 36, French (Dr. Ross)	September 15, 5 p.m., shell	September 18, 5 p.m.: T. 100° F., P. 120; poor condition; multiple wounds of buttocks and left thigh, left leg and ankle; fracture of right ischium; much gangrenous muscle in thigh wounds; left peroneal muscles gangrenous, very bad odour right buttock. September 26: Diarrhœa; T. 104° F., P. 130; ? septicæmia	September 19: Fractured bone removed from buttock; one in right ischium firmly imbedded; *capote* removed from all wounds; gangrenous muscle and skin cut away; salt and carbolic dressing. September 26: Further operation, sequestrotomy	*Bacillus perfringens*, staphylococcus, streptococcus	*Bacillus perfringens*	Not examined	Shell in thigh; extensive comminution of right ischium	13 days; death, September 28, 7 p.m. (first dressing, September 16, shell removed). Post-mortem: Retroperitoneal muscles dark in colour and congested, but not gaseous; much swelling; clinically septicæmia

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
93	C., 24, French (Dr. Nicholson)	August 26, 8 p.m., shell	August 30: T. 99° F., P. 82; wound in midline of left calf, tense, and very painful; discoloration of skin, gangrenous tibial muscles; gas; healed well	August 30: Muscle cut away; fractured bone removed; deep muscle gangrenous	Staphylococcus, scanty *Bacillus perfringens*, pus, streptococcus	*Bacillus perfringens*	—	(I) Shows swelling of calf; fractured bone behind tibia; deep gas tracery; early stage of gangrene; August 30, 1916	37 days; evacuated October 2. Classic form (localized). On admission wound stitched up
94	L., 22, French (Dr. Ross)	July 20, 6 a.m., shell	July 23: T. 100° F., P. 100; good condition; large dirty wound on outer and posterior aspect of right thigh, exposing great trochanter and anterior wound in groin external to vessels; foul discharge, gas. July 24: Much swelling of abdominal wall and round wound, much serosity. July 26: Swelling subsided	July 23: Much gangrenous skin and muscle cut away; sequestrotomy. July 27: Great trochanter removed; 1 in 1,000 iodine; salt and carbolic; Saissi	Diplococci, *Bacillus perfringens*, *Bacillus coliformis*, ? *Bacillus œdematiens*	Streptococci *Bacillus perfringens*, *Bacillus sporogenes*, diplococci* *Bacillus œdematiens**	July 27, sterile	(I) Comminuted fracture of neck and great trochanter right femur; gas infiltration, extreme; July 23. (II) Callus formation. October 19, 1916. R. 94	4 months; November 3: Wounds superficial. (First dressing 16 hours after wound.) Œdematous form, which yielded to serum treatment
					July 23: 11 p.m., 40 c.c. anti-œdematiens serum, 10 c.c. anti-perfringens serum. July 26: 6 p.m., 40 c.c. anticœdematiens serum, 40 c.c. antiperfringens serum.				
95	L., 21, French (Dr. Nicholson)	July 8, shell	July 14: Large penetrating wound of right iliac region, gangrenous muscle; extensive fracture of head of femur and pelvis. August 1: Anæmia, temperature rising	July 14: Wounds cleaned. August 1: Excision of head of femur; drainage. November 20: Sequestrotomy	Streptococcus, staphylococcus, *Bacillus perfringens*	*Bacillus perfringens*, *Bacillus sporogenes*	Sterile	Fracture of pelvis, right femur	4½ months. 2·5 c.m. shortening. (Drainage at V——.) Early stage of œdematous form

96	A., 21, French (Dr. Ross)	September 15, shell	September 18: T. 99° F.; offensive wound between outer border of right scapula and vertebral column; muscles dark, infected	September 19: Excision of gangrenous muscle; 1 in 1,000 iodine	*Bacillus perfringens*, *Bacillus sporogenes*	*Bacillus perfringens*, *Bacillus sporogenes*, aërobic spores	—	—	21 days. September 30: Healing well. October 5: Evacuated, healing. No complications. (September 17: Shell removed.) Early classic form localized
97	Q., 41, French (Dr. Martland) (*see* fig. 10, p. 74)	September 12, 4 p.m., shell	September 18: T. 103° F., P. 126; small dirty wound on right calf, outer aspect near head of fibula; leg and foot swollen and œdematous, not cold nor discoloured; much swelling and discoloration in popliteal space; offensive discharge; thrombosis of popliteal vein; external popliteal nerve injured	September 19: Operation, gangrenous muscle semimembranosus cut away; shell removed and through drainage established	Staphylococci, streptococci	September 23, *Bacillus perfringens*, *Bacillus sporogenes*, staphylococci, streptococci	—	September 19, 1916. (I) No fracture; large projectile behind femur in popliteal space; cloudy area adjacent; ? pus or gas	2 months. November 17: Nearly healed. Early localized classic form
98	N., Arab (Dr. Hodson)	July 6, shell	July 7: T. 101° F., P. 112; collapsed, toxæmic; perforating wound through left gluteal muscles to left adductor region; pain in left groin; adductor longus and part of brevis gangrenous; red offensive discharge	July 8: Wounds opened up, entrance and exit; connected by incisions; gangrenous muscle cut away; iodine 1 in 1,000	Abundant *Bacillus perfringens*	*Bacillus perfringens*, streptococcus	—	No fracture	6 weeks. August 15: Clean. Evacuated, August 18, healing. No complications. Early localized classic form
99	S. D., 21, Senegalese (Dr. Courtauld)	July 1, shell	July 2: Septicæmic; T. 98° F., P. 120; foul smelling, perforating wound of left buttock exuding gas and brown discharge: buttock immensely swollen; rectum perforated, but not completely divided; gangrenous muscles. July 4: T. 97° F., P. 140; toxæmic condition; very foul odour	July 3: Operation; wound opened up widely	—	*Bacillus perfringens*, *Vibrion septique*, *Bacillus œdematiens*, streptococcus	Culture not made	Fracture of sacrum and coccyx	5 days; death, July 6, 5 a.m.; œdematous form; in a hopeless condition on admittance

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
100	P., 27, French (Dr. Ross)	September 4, 7 p.m., shell	September 17, 11 p.m.: Collapsed; T. 100° F., P. 130; gangrenous multiple wounds in both scapular regions; maggots on dressing, very infected; shell in left lung, no gas, but very foul pus, not much swelling; head of humerus infected, shell perforating body of scapula and lying in infected subscapularis muscle. September 19: T. 103° F., P. 104. October 30: Healing well, arm in good position	September 7: Shell removed. September 18: Wounds cleaned; gangrenous muscle cut away, *capote* removed. October 2: Head excised, drainage by Saissi's tube. October 19: Shell removed from sub-scapularis through axillary incision; Thomas's splint	Severe mixed anaërobic infection	*Bacillus perfringens*, *Bacillus sporogenes*, *Bacillus œdematiens*, streptococcus	—	(I) Shell; compound fracture of glenoid cavity of left scapula and of left head of humerus; fracture of right scapula; September 18, 1916.	Local œdematous type. October 30: Healing; right arm in good position
101	B., 20, French (Dr. Wilson)	October 21, 1915 shell	October 24, 1915: T. 103° F., P. 120; fair; very dirty wound of left leg, anterior and internal surface; tibia fractured and bone marrow exposed; foul smell; did not clean. October 30: Leg began to swell, with T. 103° F., P. 130; yellow. November 6: Signs of pneumonia at left base; anæmic; T. 103° F., P. 120; T. swinging for weeks; gradual improvement	October 24: Incisions; wound cleaned, much *capote* removed. October 26: Incisions. October 29: Incisions; saline drip. October 30: Amputation above knee-joint; salt and carbolic. March 6: Re-amputation	—	*Bacillus perfringens*, streptococci, *Bacillus coliformis*	No blood culture made, probably a streptococcus present	(I*a*) Compound fracture of left tibia; shows deep gas infiltration. (I*b*) Shows gas infiltration (linear) in subcutaneous tissues	198 days; evacuated healed, May 7, 1916. Latent œdematous type. Infection probably kept up by retained *capote*
102	B., 25, French (Dr. Wilson)	January 29, 1 p.m., shell	January 29, 1916: T. 102° F., P. 80; exhausted; right arm red, swollen from wrist to upper (mid) arm; large, foul, sloughing wounds with gangrenous muscle and cut tendons protruding in middle of right forearm; penetrating wounds of right arm, chest, shoulder and back. January 31: Much swelling and foul discharge from wounds of arm; arm swollen to chest, skin discoloured. February 2: Pus bright yellow	January 30: Sequestrotomy. January 31: Circular amputation right arm with lateral incisions; skin incisions in pectoral region; salt and carbolic. March 18: Re-amputation, drip, salt and carbolic dressing	—	*Bacillus perfringens*, *Bacillus subtilis*, *Bacillus proteus*, streptococcus, *Bacillus sporogenes*	—	(I) Fracture of right ulna and radius. (II) Right upper arm shows gas bubbles near shell; January 30, 1916	99 days; evacuated healed, May 7, 1916. (First dressing 3 hours after wound.) Œdematous type

103	R., 20, French (Dr. Wilson)	January 27, 7 p.m., shell	January 29, 1916: Multiple wounds of scalp, right antrum, left arm and hand, right forearm and hand, both knee-joints, left buttock, thigh, and left calf; red fluctuating crepitant swelling left gluteal fold. January 31: Very talkative, conscious; foul gaseous pus from left buttock and thigh, incontinence of urine, thin brown discharge. February 1: Colour bad, very yellow; foul smell; breathing rapid; left leg crepitant, and swollen to hip, and spreading rapidly to thorax; large bullæ filled with foul, dark green fluid; abdomen swollen; hæmorrhagic vomiting; 12 midday, death; conscious almost to end	January 30: Operation; many fractured bones removed; foul gas abscess in left thigh opened and *capote* removed; abscess in left buttock containing gas opened and drained; salt and carbolic dressing	—	*Bacillus perfringens*, streptococcus, staphylococcus	Probable septicæmia, no blood culture	Multiple fractured bones; fracture of right upper jaw. (I) Print shows shell in adductor region. January 30, 1916	6 days; death, February 1; classic form
104	B., 33, French (Dr. Nicholson)	September 25, 1915, grenade	September 26, 1915: Fatigued; T. 100° F., P. 120; huge, gaping wound on front of right thigh, offensive discharge; extensor muscles severed transversely; much pain in wound; gas exuding	September 26; Cleaned, sloughs cut away; 5 per cent. salt and carbolic dressing	—	September 28, *Bacillus coliformis*, *Bacillus sporogenes*, streptococcus, *Bacillus perfringens*	—	No fracture	48 days; evacuated healing, November 11, 1915; local classic type
105	B., 19, French	October 10, 3 p.m., shell	October 10, 1915: T. 100° F., P. 120, R. 30; hæmoptysis; great pain in right leg; right leg wounded, discoloration of skin over tibia, sloughs of muscle; deep cavity in tibia filled with foul pus; œdema of dorsum of foot and lower part of leg; some gas; septic wound of right hand; penetrating wound of right lung (shell). October 12: Much gelatinous œdema. October 15: Swinging temperature, flatulence. November 9: Empyema, signs of septicæmia; T. 103° F., P. 132. January 23: Suppuration in stump; good recovery	October 10: Fractured bone removed from abscess in leg; drainage of chest and hand. October 12: Drip; incisions. October 14: Amputation lower third thigh. November 9: Empyema drained. January 12: Shell removed, hand. January 22: Re-amputation. April 10: Scar freed	—	*Bacillus perfringens*, streptococcus, Coliform bacillus *Bacillus pyocyaneus*	—	(I) Shell seen in leg; extensive gas tracery; October 10, 1915	210 days; evacuated healed, May 8, 1915. Œdematous form

No. of Case	Name and age	Date of wound; projectile	Date of admission and general condition	Treatment	Bacteriological Report			X-ray examination; date of plate	Duration of illness; result; remarks
					Smear	Culture	Blood		
106	M., 44, French (Dr. Nicholson) (*see* fig. 13, p. 77)	October 7, 2 p.m., shell	October 16, 1915: Foul smelling wound of left leg and on anterior aspect of lower third of left tibia; ends of bones seen. October 26: Wound very dirty	October 16: Drained, cleaned, drip. October 26: Amputation lower third thigh: drip. January 14: Re-amputation middle third	—	October 16, *Bacillus tetanus*, *Bacillus perfringens*, *Vibrion septique*, streptococcus, *Bacillus proteus*	—	(I) Compound fracture left tibia, with loss of substance and marked striation. October 16, 1916.	187 days; healed; evacuated, April 12, 1916; œdematous form (latent)
107	C., 32, French (Dr. Robinson)	October 23, shell	October 26, 1915: T. 98° F., P. 100; apparently slightly infected wound of left calf; foul smell, gas. October 27: Swelling in calf, hæmorrhage. October 28: Foot cold and pulseless, T. rising; gas crackling in calf; T. 98° F., P. 140. October 30: Much swelling of stump; complains of pain in chest; signs of gas septicæmia; hæmorrhagic vomiting; gas bubbled continuously from stump; enormous swelling	October 26: Shell removed; drainage. October 27: hæmorrhage from posterior tibial; traumatic aneurysm of calf, popliteal artery ligatured. October 28; Amputation of thigh, upper third; salt and carbolic	—	*Bacillus perfringens*, streptococcus, staphylococcus	—	Projectile in left leg	8 days; death, October 31, 8 a.m.; classic form

Section of Surgery.

President—Surgeon-General Sir ANTHONY A. BOWLBY, K.C.M.G.

(*March* 14, 1917.)

(Chairman—Lieut.-Col. H. J. WARING, R.A.M.C.)

The Treatment of Severe Infected Gunshot Injuries of the Shoulder- and Elbow-joints; Early Excision to secure Mobility.

By R. H. JOCELYN SWAN, M.S.Lond., F.R.C.S.Eng.,
Major R.A.M.C.

THE treatment of gunshot injuries to important joints, complicated by extensive comminution of the articular surfaces and by septic infection, has received considerable attention, particularly in regard to the knee-joint, where the anatomical conformation of the synovial membrane offers such difficulty in securing adequate drainage. In a large number of severe injuries of the knee the best that can be hoped for, without amputation, is an ankylosed joint, and the patient is left with a good prospect of earning his living. A fixed shoulder or elbow, however, causes much greater physical disability as far as the earning capacity of the individual is concerned, and it is with the object of obtaining a movable and useful joint that I have adopted a method of early excision of the joint with such satisfactory results that I am tempted to put them together, with the hope that they may prove of assistance to others.

The ultimate results obtained in the earlier period of the war after extensive injuries of both the shoulder- and elbow-joints were by no means satisfactory, drainage of the joint resulting in fixation of the

articular surfaces, whilst sinus formation from necrosing fragments of bone often necessitated further operative measures before healing was obtained. Fixation of the shoulder, with inability to abduct, flex, extend, or rotate the arm, is a serious disability, whilst any fixation in the elbow at an angle about 80° renders the arm useless for feeding, and of very limited use for mechanical purposes. In my earlier cases I considered it advisable to leave the joint alone after free drainage and removal of any obviously loose fragments of bone until firm healing was obtained and then proceeded to a formal excision of the joint, but I found so much matting and infiltration of the ligaments, muscles, and structures in immediate relation to the joint from the previous septic inflammation, that not only was the operation rendered difficult, but the results obtained were not satisfactory. In addition, the time occupied in the healing of the original wounds, the liability to localized necrosis requiring local operations, and the pain experienced, were a severe tax on the patient's strength, and he was often inclined to refuse further operative procedure.

In reviewing a large number of cases of gunshot wounds of the shoulder- and elbow-joints they may be roughly classified as: (1) Penetrating wounds without fracture or sepsis; (2) penetrating wounds without fracture but with sepsis; (3) fracture of the articular surfaces without sepsis; and (4) fracture of the articular surfaces with sepsis.

Each of these classes may be further complicated by the presence of a bullet or metal fragment, imbedded in the ends of the bone, in the joint, or in the tissue surrounding the joint. In the majority of cases the bullet or fragment conveys infection with it from particles of earth or clothing accompanying it, and the question of removal must be decided upon in each individual case from the amount of trouble caused.

The treatment of the joint depends as much upon the degree of sepsis arising as upon the anatomical injury produced. Where no sepsis is present in the joint the wounds of entry and exit may be excised but not sutured, the joint fixed on some suitable splint and treated on general surgical lines. Personally I must say that I regard the excision of primary gunshot wounds of a limited or even doubtful advantage, and have seen many cases of bad results from this practice. Where septic infection has been conveyed to the interior of the joint more active measures must be adopted, and the joint cavity thoroughly irrigated.

I would here note the difficulty in determining the presence of septic infection within the joint. It has been frequently observed that the joint may be distended with pus containing streptococci, coliform organisms, and *Bacillus perfringens*, but yet there is comparatively little pain, and the temperature and pulse-rate are only slightly raised—in fact, a very different clinical feature to the acute septic arthritis of civil practice. With a gunshot wound penetrating a joint, one may be tempted not to open the joint in the absence of constitutional disturbance, whereas delay where sepsis exists may make all the difference in the ultimate functional utility of the joint. An inflamed area around the entrance or exit wound, the discharge of thin mucoid fluid on pressure, commencing thickening of the tissues around the joint, a slight elevation of temperature, and a commencing leucocytosis are indications of infection and of the necessity of exploration and irrigation.

It is, however, to cases of extensive bony injury to the articular surfaces, associated with a virulent infection, that I desire to draw attention. These injuries are so extensive, and appear at first sight so hopeless, that the limb may be amputated, whereas if saved at the expense of a prolonged convalescence after free drainage and the removal of necrosing fragments of bone, the resulting fixation of the injured joint is a source of great physical disability to the patient. The septic infiltration of the tissues surrounding the joint, together with the filling up of the actual fossæ in the bone around the joint by callus during the period in which the limb must be kept at rest, all tend to prevent mobility, whilst the erosion of the articular cartilage in the pathological processes leads to absolute bony fixation.

I was faced with this difficulty in the case of a young soldier whose right arm had been amputated immediately below the shoulder-joint, and whose left elbow-joint was extensively smashed and septic. In this case fixation of the elbow at such an angle as to allow him to feed himself would have left him with an arm of little use for any other purposes, whilst fixation at 120° to 150°, to give him the greatest mechanical advantage in the limb, would have rendered it useless for feeding. After much consideration I decided to excise the damaged elbow-joint freely, even in the presence of marked sepsis, leaving the cavity freely open, and after three months this patient could flex and extend his elbow to a range of nearly 90°, and had excellent muscular power in the arm—in fact, he surprised us by lifting and flexing the elbow whilst holding a heavy scuttle of coals. This case gave me much food for reflection, and I have since adopted the principle which I

now advocate—namely, free and early excision in cases involving severe comminuted fracture of the articular surfaces of bone, complicated by septic infection, with the direct object of obtaining a movable joint.

The Treatment Adopted.

Cases are received at Woolwich at varying periods from the date of injury, but it is no uncommon thing for them to arrive within three days of being wounded. In all septic cases I make a routine practice of giving an injection of a mixed polyvalent streptococcus and proteus vaccine, as I have described elsewhere,[1] and the wounded area is carefully skiagraphed to determine the nature and extent of any bony injury and the presence of metal fragments. I consider this preliminary injection of "antisepsis" vaccine of as much importance as the prophylactic injections of antitetanic serum, as by its means the spread of anaërobic infection is limited.

If the ends of the bone entering into the formation of the shoulder or elbow-joint are severely fractured, and if there is evidence of septic infection in the joint, I do not hesitate to open the latter freely, remove all completely detached fragments, and saw off the ends of the bone; the whole cavity is flushed with a forcible stream of hot sterile water, and after arresting the bleeding, gauze is very lightly inserted into the cavity, into which are brought three or four small perforated tubes attached to a reservoir containing Dakin's solution or hypertonic saline, by which the cavity is frequently flushed. The incision is left freely open, perhaps one suture being used at the upper end. In the case of the shoulder I make use of an oblique anterior incision between the deltoid and pectoralis major muscles, opening the joint at the side of the long tendon of the biceps. Frequently the actual damage to the bone is more severe than the radiograph would indicate, the head may be completely separated from the neck and displaced. If possible, the tuberosities and rotator tendons are left intact, but if the fracture involves the tuberosities they are removed, the periosteum being stripped from the upper part of the shaft and the bone divided through the surgical neck. A stab incision is then made from the posterior aspect of the joint backwards to come out behind the shoulder, or the bullet track used if conveniently placed and a drainage-tube inserted, the flushing tubes being introduced by the anterior incision. The arm is

[1] *Lancet*, 1916, ii, p. 859.

then fixed in a fully abducted and slightly flexed position, and in my later cases I have made use of a special splint I have had made for me by Messrs. Allen and Hanbury for both these cases and for gunshot fractures of the upper third of the humerus, extension in the humerus being maintained from the flexed elbow. The arm rests very comfortably in this splint, the wound can be easily dressed without removing it, and the patient is able to be up and walk about; the deltoid muscle is maintained relaxed and does not show the same degree of wasting as when the arm is fixed to the side.

In the elbow I use a free, median, posterior incision, separating the tissues close to the bone and as far as possible preserving the triceps tendinous attachment, and, of course, the ulnar nerve. The lower end of the humerus is projected from the wound and removed as may be found necessary, usually above the epicondylar line. The olecranon is removed together with the upper cartilaginous surface of the coronoid process and the cupped disk of the head of the radius. The cavity is then flushed with hot sterile water, lightly filled with gauze, and the irrigation-tubes introduced. The arm is put up, flexed, and supinated, the wound being left freely open. After a few days, when granulations appear on the surface, passive movements are commenced and the forearm put up in a different angle of extension each day. As the wound fills up voluntary actions are encouraged.

In two cases of severe fracture into the elbow I have been content with the removal of the fractured humeral condyle, leaving the olecranon and radius untouched. In each case the internal condyle and trochlear surface was removed at an early stage and the joint treated as in a complete excision. At the operation cultures are taken from the interior of the joint and a full bacteriological examination made, from which specific autogenous vaccines are prepared and given at intervals of four or five days, under the direction of my colleague, Mr. Kenneth Goadby, who will detail the bacteriology of the cases.

It might be noted here that the bacteriological findings in these cases show the presence of not only pyogenic cocci and bacilli of the coli group, but also *Bacillus perfringens*, of malignant œdema, and *Hibler IX*. The operations in practically all the cases were done in the presence of active and virulent sepsis, but by treatment with free drainage, irrigation and vaccines the sepsis was at once controlled. There is usually a slight elevation of temperature for a few days after operation, but it soon settles down, and in only one case have I had to incise the limb after the operation for any spread of sepsis.

Results.

So far I have operated in an early stage upon six cases for excision of the shoulder-joint, and on eight for excision of the elbow-joint in cases of severe comminution of the articular surfaces associated with sepsis.

Shoulder-joint Cases.—One remains in hospital, but it is doing well; of the remaining five, in one a sinus remained after seventeen weeks due to the necrosis of some small fragments which were removed. Of these five cases the ultimate result was good in two, free movement of flexion and extension and abduction without scapular movement to an angle from 50° to 60° with the vertical being obtained. In two other cases the result was fair, good flexion and extension, but abduction was limited to 40° with the vertical, whilst in the remaining one there was evidence of circumflex nerve injury from the primary wound which persisted, so that abduction was impossible owing to the paralysis of the deltoid, and the patient refused further operative treatment to deal with the nerve lesion. It is interesting to note that in cases of excision of the shoulder, where extensive removal of the bone through the surgical neck was performed by subperiosteal resection, subsequent radiographs demonstrated that there was considerable re-formation of bone in the line of the shaft of the humerus towards the glenoid cavity.

Elbow-joint Cases.—Here the ultimate results have been even more satisfactory. Of the eight cases operated upon one is still in hospital from operation too recent to include as a result; of the remaining seven cases, one required further operation on the ninth day from spread of sepsis in the middle of the arm. Of these seven, in four the ultimate result exceeded all my expectations, flexion and extension being possible to almost full extent and supination and pronation being possible, but not perfect; all show some degree of lateral mobility, but that does not interfere with the usefulness of the limb, and the muscular power in the arm and forearm is excellent. In each of these four cases, flexion of the elbow is so good that the patient can voluntarily touch the shoulder of the same side, can fix a collar-stud at the back of the neck, and can touch the angle of the scapula of the opposite side; in each extension is possible to almost a straight line. Each one could lift and raise a fairly heavy ward chair with the elbow flexed and unaided. In two cases flexion and extension are possible in a smaller range, in one from 80° to 145°, and in the other from 80° to 120°, further movement being limited by masses of callus formed about the ends of the bone. In the latter

case the operation for excision was performed at a later period after free drainage had proved ineffectual to arrest the infective processes. In the remaining case, in which the upper fourth of the radius had to be removed owing to the excessive shattering, flexion and extension are possible in a range of 45°, but no movement of pronation or supination is allowed, owing to the absolute fixation of the upper end of the radius to the ulna by a large mass of callus joining the two bones.

In addition to the above I have excised two hip-joints for extensive fracture of the head and neck of the femur with septic infection in the joint. One case did well, and the patient walks with a limb shortened by 1¼ in., but with good movement at the hip. The other case was doing well, but died suddenly on the thirteenth day from a profuse hæmorrhage from a chronic duodenal ulcer which opened the pancreatico-duodenal artery. So far I have had no opportunity of excising a knee-joint in a recent case of gunshot injury, but in the light of other joint cases and with my experience of the difficulty of adequately draining a septic knee-joint, I would not hesitate to do so, but in this instance with the distinct object of obtaining a firmly ankylosed joint.

I am of opinion that the earlier the operation of excision is performed the better is the chance of obtaining free mobility. In my cases the best results have followed operations performed soon after the injury. Naturally a very great deal of the success of the operation depends upon the assiduity of the after-treatment and the hearty co-operation of the patient in carrying out early movements, and I would here like to express my sincere thanks to the various medical officers who have had charge of these cases and who have so faithfully carried out my instructions.

Advantages of the Operation.

(1) All loose fragments of bone and the articular surfaces are removed, having a clean sawn surface rather than jagged ends of bone.

(2) Very free drainage is established, and the joint can be freely irrigated.

(3) The septic processes are at once controlled and pain is much diminished. The chance of having to amputate for advancing sepsis is much lessened.

(4) The time of convalescence is very much diminished and there is much less risk of localized necrosis of bone and sinus formation.

(5) A movable joint is obtained with good muscular powers.

Objections to the Operation.

(1) *Shock.*—There is necessarily more shock in an excision of a joint than in making free incisions for drainage, but actually the shock is very slight. The operation can be easily performed in half an hour, bleeding is easily controlled, and no large nerves are divided as in amputation. I have seen no necessity for amputation having to follow in any case from failure to control the sepsis, but, should it become necessary, the previous excision of the joint would probably increase the risk. There has been no instance of secondary hæmorrhage.

(2) The fact that *freshly sawn surfaces of bone are exposed to septic infection* might seem to be a disadvantage, but with the free drainage, intermittent flushing of the cavity, and the use of vaccines the risk of septic osteomyelitis appears to be small.

(3) The risk of a *flail joint resulting.* This is a possibility which cannot be overlooked, especially in the elbow. I can only say that I have not seen it in my cases, and I have removed so much bone that three fingers could easily be placed between the sawn ends with the forearm extended, but I have seen one case in which the joint was flail after an excision of the elbow performed at a later stage.

(4) The excessive formation of callus around the divided ends of the bone may limit free movement. In cases in which the comminution of bone is excessive, or where splinters or minute particles of bone are conveyed into the surrounding tissues, this formation seems to be more excessive and may limit free movement; but where movement is commenced early, and the patient steadily perseveres with it, good range of movement is obtained. In some cases it is surprising that such good movement is obtained where large masses of ossified callus surround the ends of the bone.

It may be justly urged on theoretical grounds that it is unjustifiable to remove so much bone in the presence of active and virulent sepsis, or that I am urging a procedure which contravenes ordinary surgical principles, but I can only point out the success that has attended these operations. I am conscious that the number of cases is small, but I have not included those in which the operation has been performed when the wounds were healed and the joint fixed, or those in which the operation was done at a later stage when necrosis of the end of the bone had commenced. However, I feel most strongly that if in these cases of septic gunshot fractures, involving the joint, excision or partial excision were performed soon after the injury, we should be doing more

for the wounded man in putting him into a better position to earn his future livelihood, we should materially shorten his convalescence, and thus save the State not only many of the expenses incurred during hospital treatment, but also the amounts paid in pensions.

I must thank Colonel R. J. S. Simpson, A.D.M.S., Woolwich, for permission to make use of the cases forming the basis of this communication, and my colleague, Dr. Martin Berry, for the excellent series of radiographs used in illustrating it.

Shoulder-joint Cases.

Case I.—Private B. Wounded on December 3, 1915. Bullet entered at back of left shoulder and passed out by a large wound in front. Operation in France; anterior wound incised and shoulder-joint opened. January 3, 1916: Admitted to Woolwich. Free discharge from anterior wound, great pain on any movement of shoulder. Deltoid paralysed and circumflex anæsthesia. Skiagraph showed much comminution of head and neck of humerus; head of bone turned, so that articular surface faced outwards. January 6: Operation. Wound opened; upper end of humerus of dark-grey colour and articular cartilage removed. Periosteum separated and bone divided through surgical neck. Area flushed with hot saline and wound lightly packed with gauze with large drainage tubes. Arm fixed, abducted with extension from elbow. January 19: Wound granulating well. Temperature normal since operation. Arm fixed on Hey Groves crutch splint. No pain in shoulder. January 27: General condition much improved. February 14: Wound healed. Passive movements are free, but patient will not make any attempt towards active movement. Deltoid paralysed. March 20: Flexion and extension good. No abduction. Deltoid wasted. June 1: Can flex and extend shoulder, but no abduction. Circumflex paralysis remains. Movements of elbow and hand good. Skiagraph showed large deposition of bone in line of shaft occupying position of piece removed. No necrosis nor sequestrum seen.

Case II.—Private H. Wounded December 12, 1915, bullet entering at point of right shoulder and coming out in pectoral fold. February 27, 1916: Sinuses remain at each wound and dead bone easily felt by probe. Free discharge of thick pus. Skiagraph showed disorganization of head of humerus; articular surface pointed upwards. Rarefied area in neck, probably necrotic. March 4: Operation. Much disorganization found in shoulder-joint, the head of humerus being blackened and soft necrotic area below it. Tuberosities not involved. Section made through anatomical neck and necrotic area thoroughly scraped. Cavity flushed out. Free drainage. Arm fixed in abducted position. March 24: Slight pyrexia for first week. Wound closing rapidly. Passive movements commenced. April 20: Wound healed. Moves shoulder a little. July 13: Can flex and extend arm and abduct to 40°. Deltoid acts well. No rotation in shoulder, but movements are increasing.

Case III.—Corporal G. Wounded January 24, 1916, bullet passing through right shoulder. Two operations in France where shoulder-joint was opened and drained. March 17: Two healed wounds on outer aspect of shoulder and large open wounds both in front and behind, through which a drainage-tube passes. Great pain in any movement. No paralysis of arm. Skiagraph showed extensive comminution of upper end of humerus, the head being completely separated from the shaft, the drainage-tube passing between interval. Upper end of shaft necrotic. March 28: Operation. Anterior opening enlarged and joint opened. Head of humerus very necrosed and devoid of articular cartilage. Upper end of shaft also necrosed. Periosteum separated and shaft divided through surgical neck, 1½ in. being removed. Granulations scraped away and bleeding arrested by hot saline. Cavity irrigated with hydrogen peroxide, large drainage-tube in posterior wound and small tubes for irrigation from front wound. Light pack of gauze in wound. April 2: Very little discharge and granulations appearing. April 17: Wound healthy, passive movements commenced. May 10: Not much active movement in arm yet. July 15: Very fair movement in shoulder; flexion and extension good, but abduction limited to 40°. Skiagraph showed a new formation of bone extending in line of shaft towards glenoid cavity.

Case IV.—Private G. P. Wounded July 1, bullet entering front of left shoulder 1½ in. outside tip of coracoid process and emerging at slightly lower level behind. Admitted July 5, when a radiograph showed fracture of the clavicle, of the acromion process, and of the anatomical neck of the humerus, the head of the latter being comminuted and displaced. July 6: Wounds opened and free drainage established through joint. July 27: Temperature raised for last three days and local swelling about shoulder. Wound opened and head of humerus found blackened and necrosed. Decided to excise head; periosteum peeled back and muscular attachments to tuberosities detached. Resection through upper part of surgical neck. Joint cavity flushed with hydrogen peroxide and saline and freely drained by posterior incision. Intermittent flushing with Dakin's solution. Arm maintained abducted. September 25: Wounds all healed. November 12: Can voluntarily abduct arm to 45° from body, and on fixing scapula passive movement can be made to 80° before scapula rotates. Flexion and extension of arm good. Skiagraph showed a fair growth of callus around upper end of shaft. Glenoid irregularly thickened. January 4, 1917: Movements at shoulder are more free. Can abduct easily to 60° without rotation of scapula. Flexion and extension are freer. Result excellent. (*See* figs. 1 and 2.)

Case V.—Private H. MacI. Wounded July 1, 1916, by rifle bullet entering in front of right shoulder just outside tip of coracoid process and emerging in posterior deltoid margin. Admitted July 4. Anterior wound sloughing. Thin, serous bubbly pus draining. Great pain in shoulder. No nerve injury. Skiagraph showed very comminuted fracture of head of humerus. Articular surface rotated. July 6: Operation. Incision in pectoro-deltoid interval,

joint opened. Head of humerus much comminuted. Upper end of shaft black and necrotic. Periosteum stripped off tuberosities and detached bone and upper end of shaft removed. Area flushed with peroxide and hot saline and cavity lightly packed with gauze. Large drainage-tube through posterior wound. Arm put up fully abducted and arranged for frequent flushing. July 9: Temperature about 100° F. Wound clean and commencing granulation. July 20: Wound excellent. Very little discharge. August 22: Arm removed from splint and passive movements commenced in shoulder. Wound almost healed. November 11: Small sinus remained in incision and skiagraph showed small sequestra at upper divided end of humerus. Can move shoulder surprisingly well. Can abduct to 45° without movement of scapula. November 13: Sequestra removed. January 10, 1917: Wound healed. Movements of arm increased. Can abduct to 60° and flex and extend well. Can passively raise arm to almost vertical. Result is very good. (*See* figs. 3, 4, and 5.)

FIG. 1. FIG. 2.

Figs. 1 and 2 (Case IV).—Fig. 1, radiograph taken three and a half months after excision of shoulder, shows the ossification of callus at the upper end of shaft of humerus. Fig. 2 shows the movements obtained; note that the figure showing elevation of the arm is a passive movement, the others active and voluntary.

Case VI.—Corporal E. Wounded July 29, 1916, by shell fragment entering posterior aspect of left shoulder and emerging at tip of coracoid process. Admitted August 3. Patient sallow and sweating. Temperature 98° F. Pulse 112. Wounds foul and fine crepitation felt around anterior aspect of shoulder. Skiagraph showed great comminution of head of humerus, which is separated from shaft. Glenoid cavity and neck of scapula comminuted. Fracture of coracoid process and spine of scapula. Three fairly large metallic fragments present. Operation (on admission): Wound freely opened and direct

FIG. 3.

FIG. 4.

FIG. 5.

Figs. 3, 4, and 5 (Case V).—Fig. 3 is a radiograph of the original injury to the head of the humerus, with drainage-tube *in situ* (July 4). Fig. 4, radiograph four months after excision. Fig. 5, the movements obtained; note that the elevation of the arm is a passive movement, the others active movements.

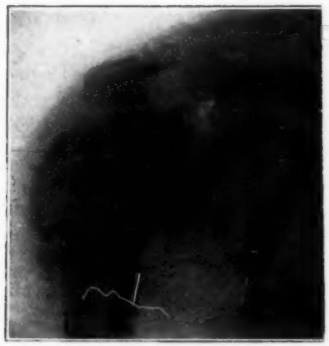

FIG. 6.

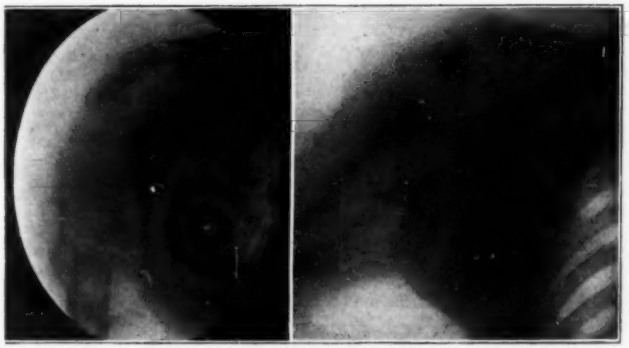

FIG. 7. FIG. 8.

Figs. 6, 7, and 8 (Case VI).—Fig. 6 shows the injury to humerus, scapula, and clavicle five days after the receipt of wounds, the central clear area being a gaseous collection in anaërobic infection. Figs. 7 and 8 are radiographs taken two months after excision, showing the movement of the mass of callus with the humerus.

communication made from front to back through joint. Bone very comminuted and very foul. Smear preparations showed numerous spore-forming bacilli. Area swabbed with peroxide and tubes arranged for continuous irrigation. Saline infusion. August 6: General condition good. Temperature 98·6° F. Pulse 96. Tongue clean. August 14: Condition unsatisfactory. Temperature up to 102° in evenings, but tongue moist. Drainage not so free. August 15: Operation. Wounds freely opened. Bone found blackened and very foul. Much comminution. Purulent granulations in medullary cavity of shaft. Latter sawn through surgical neck. Glenoid cavity only held by triceps attachment and was removed, and many rough pieces of bone removed with forceps. Area flushed with saline and hydrogen peroxide and freely drained. Arranged for continuous irrigation and arm put up abducted. August 21: Temperature normal since operation. August 30: Doing well. Arm maintained abducted to 60°. September 20: Anterior wound healing well. October 20: Skiagraph showed a lot of callus around upper end of humerus which moves with shaft. Sequestra present at glenoid and upper end of shaft. December 30: Sinuses remain at each end of incision. January 9, 1917: Sinuses opened and sequestra removed. There is a small amount of mobility at shoulder, and deltoid muscle acts. Patient remains under treatment. (*See* figs. 6, 7, and 8.)

Elbow-joint Cases.

Case A.—Private O. Wounded May 28, 1915, by shell explosion in close proximity, causing extensive wounds in right arm and about left elbow. Was operated upon within two hours and right arm amputated immediately below shoulder-joint. Admitted June 5. Amputation stump very septic, wound extending into posterior axillary fold. Also septic wound of upper left forearm over olecranon. Skiagraph: Complete shattering of upper 2 in. of both radius and ulna; very comminuted. No fracture of humerus. Large metallic fragment 3 in. above joint. June 8: General condition bad. Pulse 120. Saline enemata. Wound of forearm opened for drainage and fragment removed. June 14: Operation. Vertical median posterior incision and triceps tendon detached. Detached fragments of bone removed and upper ends of radius and ulna sawn off just above the level of tubercle of radius. Humerus not sawn off. Area flushed with peroxide and cavity lightly packed. No sutures in wound. June 20: Wound clean and granulating. July 2: Passive movements commenced in elbow. Can move fingers and hand well. August 2: Wound almost healed. Only a small range of movements allowed without pain. January 31, 1916: Can move elbow through a range of 80° and has very good muscular power. Can feed himself and touch back of occiput. There is a firm prominence in front of elbow. December: Elbow had improved until he was again injured by bomb explosion. Still has a range of movement of 60° and power is very good. Skiagraph showed large formation of callus about upper end of radius and ulna which prevents full flexion at elbow. (*See* figs. 9, 10, 11 and 12.)

Case B.—Private E. M. Wounded September 25, 1915. Wound in upper left forearm, involving median nerve, and also in inner aspect of right elbow-joint. Admitted October 14, when there was great pain in right elbow, tense swelling around the joint, and a thick purulent discharge from the wounds. Temperature 103·4° F. Skiagraph showed shattering of lower end of right humerus. No fracture of ulna or radius. October 15: Operation. Wounds of right elbow opened and found to pass directly into elbow-joint, the inner part of which was destroyed. Completely detached fragments of bone were removed, including the whole of inner condyle and supracondylar ridge and the trochlear surface of humerus. Capitellar surface was intact. Rough bone edges were rounded off and cavity flushed with hot saline and with peroxide. Wound very lightly filled with gauze. Arm put up on right-angle anterior splint. The operation wound very quickly cleaned up and granulated.

FIG. 10.

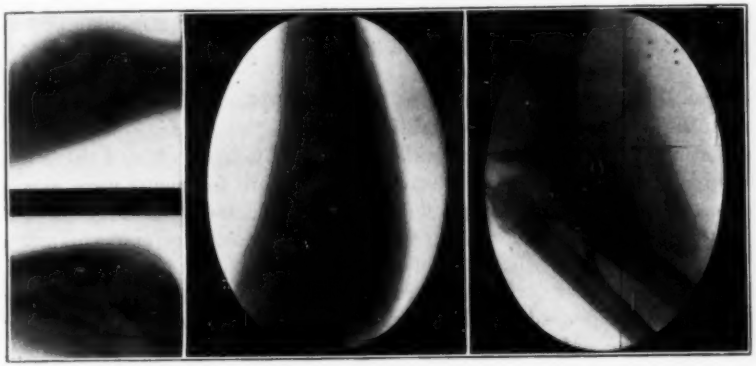

FIG. 11. FIG. 9. FIG. 12.

Figs. 9, 10, 11, and 12 (Case A).—Fig. 9 shows the great comminution of upper ends of radius and ulna, with fragments of bone driven into tissues in front of joint. Figs. 10 and 11 show antero-posterior and lateral views of the joint six months after operation (fig. 11 is upside down). Fig. 12, skiagraph after eighteen months. The large mass of callus in front of joint limited range of movement to 60°, but flexion was allowed to a right angle.

January 1, 1916: Wound healed and movements of elbow are good. January 27: Can voluntarily move elbow from 80° to 160°. The ulna has become slightly displaced inward as the result of the removal of the inner half of humeral articulation, but movement and muscular power are quite good.

Case C.—Private M. Wounded September 25, 1915, in left forearm, this fracturing radius in upper third and also on inner aspect of right elbow-joint. Arrived October 15. From the wound of right elbow there was a profuse purulent discharge; it was much swollen, extremely painful, and temperature 103° F. Skiagraph showed an extensive comminution of lower end of humerus into elbow-joint. Radius and ulna were intact. October 16: Operation. Wounds opened and passed directly into elbow-joint; the inner condyle of humerus was shattered. All loose bone removed, including the whole of trochlear surface of humerus, internal condyle, and part of the internal supracondylar ridge. The capitellar surface was left, together with the olecranon and head of the radius, which were not fractured. All rough edges of bone chipped away, cavity flushed with hot sterilized water and hydrogen peroxide. Gauze lightly packed in and two small tubes passed in for intermittent flushing. October 27: Elbow maintained flexed. Wound clean and granulating. November 23: Wound nearly healed. Passive movements in elbow good. January 1, 1916: Wound of elbow healed. Can flex voluntarily to 75° and extend to about 150°. June 27: Movements of elbow from 70° to 150°. Muscular power is very good. Ulna is slightly displaced inward.

Case D.—Sergeant S. Wounded April 21, 1916, shell fragment striking on tip of olecranon of right arm. Operation in France, where large metal fragment and several pieces of bone were removed. On admission on May 1 there was a rounded opening over point of olecranon where bare fragments were exposed. No exit wound. Elbow very painful, and there was undue mobility forward of joint. Temperature 99° F. Skiagraph showed whole of olecranon comminuted into small fragments. Fracture of lower end of humerus separating internal condyle and trochlear surface from rest of bone, extending into joint and displaced slightly forwards. May 6: Operation. Vertical posterior incision through triceps. Many small fragments of olecranon removed and pus found in joint. Base of olecranon sawn through. Humeral fracture was in good position and so a complete excision was not performed. Counter opening made on outer side and tubes arranged for continuous irrigation. June 8: Temperature gradually settled down. Very little movement allowed in elbow. July 6: Has had a lot of pain in elbow and œdema around it. Temperature 99° to 100° F. July 8: Operation. Incision reopened and lower end of humerus found to be necrotic. Further opening on inner side and joint irrigated. July 20: Temperature 101° and much purulent discharge. Operation. Joint fully opened and cartilage found much eroded. Complete excision removing humerus above intercondylar line, base of olecranon, and head of radius. Light pack of gauze and small tubes for irrigation. August 1: Doing well, wound clean. Temperature normal. August 30: Wound healed. Can voluntarily flex elbow. September 20: Can flex and extend from 80° to 120°. Supination and pronation fair. October 30: Much improved. Can touch shoulder of same side. Supination and pronation improved. Muscular power good.

Case E.—Private F. Wounded July 1, 1916, by bullet entering outer and anterior aspect of right arm 2½ in. above elbow and out over external condyle. On admission, July 5, both wounds were inflamed and surrounding tissues swollen. Great pain in moving elbow. Temperature 98·6° F. Skiagraph showed fracture of lower end of humerus, extending into joint. Much comminution, especially of external condyle. Radius and ulna intact. Several small pieces of bone in tissues in front and behind joint. July 9: Elbow very painful. Temperature 99·6°. Leucocyte count 13,500. July 11: Operation. Median vertical incision and pieces of bone removed from triceps. Joint freely opened and fracture found much more extensive than radiograph suggested, the whole of external condyle and capitellar surface and outer half of trochlear surface being completely detached, comminuted, and rotated out-

FIG. 13. FIG. 15.

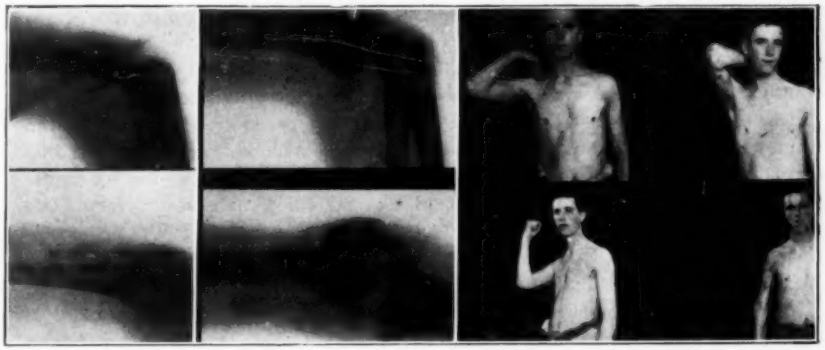

FIG. 14. FIG. 16. FIG. 17.

Figs. 13, 14, 15, 16, and 17 (Case E).—Figs. 13 and 14 are lateral and antero-posterior radiographs taken four days after injury. Figs. 15 and 16 are taken ten weeks after excision of elbow; note lateral formation of callus and slight internal displacement of forearm. Fig. 17 depicts the voluntary movement obtained.

wards. Section of humerus made ¾ in. above upper limit of olecranon fossa and olecranon removed at base, leaving cartilage of coronoid process and head of radius intact, owing to the extent of removal necessary in humerus. Cavity flushed with hot water and upper part of incision and triceps sutured. Free opening left below and tubes put in for intermittent flushing. July 20: Spread of sepsis in arm. Focus opened. July 24: Temperature not raised since last operation. Passive movements commenced. Wounds clean. August 23: Wound almost closed. Voluntary movements increasing. September 8: Wound healed. Can flex elbow enough to feed himself and to light a

cigarette. September 20: Can voluntarily flex to 80° and extend almost fully. Supination and pronation very good. December: Can flex to 75° and extend almost to straight line. Muscular power very good. Excellent result. (*See* figs. 13, 14, 15, 16, and 17.)

Case F.—Private A. M. Wounded July 29, 1916, bullet entering over tip of right olecranon and emerging at side of inner mid-line of elbow-joint. On admission, August 3, there was much ecchymosis of arm and forearm and swelling around the elbow-joint. Wounds small and clean. Temperature 98·4° F. Movements of elbow painful. Skiagraph showed very extensive damage to lower end of humerus, both condyles being fractured from shaft into joint and separated by lower end of shaft. Olecranon process also fractured. August 6: Joint more swollen. Temperature 99·8°. Leucocyte count 15,000. August 7: Operation. Posterior wound excised. Vertical median posterior incision of joint opened. Olecranon found fractured, together with a large triangular piece of upper shaft of ulna, which was removed. Both humeral condyles detached and articular surface of humerus comminuted, the lower end of shaft impinging on ulna between the fractured condyles. The muscular attachments were separated from condyles and later removed and lower end of humerus sawn off. Olecranon and coronoid processes of ulna and head of radius removed. Area well flushed with hot water and bleeding arrested. Each end of wound sutured and large median opening left for irrigation tubes. August 14: Temperature normal since operation. Very little discharge. Small drainage-tube only in cavity. August 21: Wound granulating. Passive movements begun. September 12: Can voluntarily flex elbow to 90°. September 29: Movements increasing, but muscular power poor. November 20: Movements very much improved. Can easily touch shoulder of same side, back of neck, and scapula of left side. Can extend to practically straight line. Can supinate but cannot completely pronate. Some lateral movement in elbow. Muscular power good. An excellent result. (*See* figs. 18, 19, 20, 21, and 22.)

Case G.—Private E. A. Wounded September 10, 1916, bullet passing through upper and outer part of right forearm just below elbow-joint. Admitted September 27. Wounds dirty and swelling about upper arm. Great pain on any attempted movement of elbow. No nerve injury. Skiagraph showed a very comminuted fracture of head, neck, and upper shaft of radius. Many small pieces of bone driven into surrounding tissues. No fracture seen in ulna or humerus. With such a comminution of radius and with sepsis in the elbow-joint, I deemed it advisable to remove the upper end of radius and so ensure drainage and also to attempt to obtain mobility in the elbow. September 29: Operation. Vertical incision through posterior wound. Radial head found loose and in small pieces, the articular ligament being destroyed. Many completely separated pieces of bone were removed and the sharp upper end of shaft of radius cut off with forceps. The elbow and superior radio-ulnar joints were fully exposed and flushed with hot saline and with hydrogen

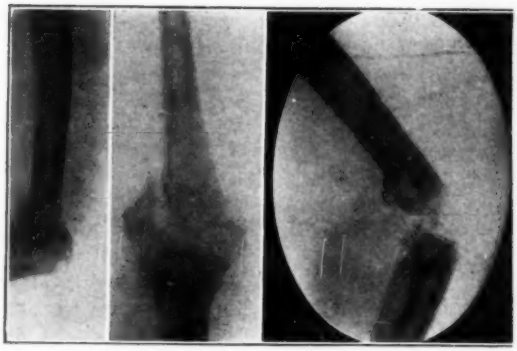

FIG. 18. FIG. 19. FIG. 20.

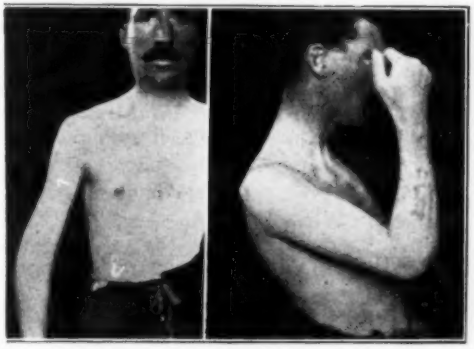

FIG. 21. FIG. 22.

Figs. 18, 19, 20, 21, and 22 (Case F).—Figs. 18 and 19 show the fracture five days after injury; note the comminution of lower end of humerus. Fig. 20, six weeks after excision. Figs. 21 and 22, voluntary movement six weeks after excision; these movements increased in range to those in fig. 17 (Case E) after a further interval of seven weeks.

peroxide. Area lightly packed with gauze with tubes for drainage. October 20: Wounds granulating well. Temperature normal. December 14: Wounds healed. Can voluntarily move elbow from 75° to 120°, and this will probably further improve. There is no movement of pronation or supination and a skiagraph shows a large mass of callus around the upper end of the radius, fixing it to ulna, which now shows a small linear fracture in same area. Probably the excessive formation of callus is due to the great comminution of bone forced into the tissues by the "explosive" force at the original injury.

Case H.—Lieutenant P. P. Wounded September 15, 1916, by shell fragment on inner and posterior aspect of right elbow. Has had five operations in France, but does not know what was done. Admitted November 14, when arm and forearm were fixed in a straight Thomas's splint, with extension. There was a large granulating surface on inner and posterior aspect of right elbow and much loss of tissue. Complete ulnar anæsthesia and paralysis. Skiagraph showed internal condyle of humerus was missing and a fracture-line through humeral articular surface separating trochlear from capitellar surface. Olecranon was fractured at base and displaced, and there was much bone débris in tissues. November 20: Temperature has been raised and more swelling around joint. Wounds opened and articular surface found devoid of cartilage, roughened and eroded. Counter openings made for free drainage. December 12: Skiagraph showed necrosis was extending. December 15: Operation. Vertical median posterior incision. Olecranon removed. Humerus projected from wound and $1\frac{1}{2}$ in. of lower end removed, but not up to limit of fracture-line on inner side. Upper end of ulna and radius was necrosed, and so removed by saw through base of coronoid process and head of radius. Bleeding arrested and area well flushed with hot water, and tubes arranged for intermittent flushing with Dakin's solution. There was $1\frac{3}{4}$ in. interval between sawn surfaces when forearm extended. December 20: Temperature normal since operation. Wound flushed very frequently with Dakin's solution, alternating with hypertonic saline. December 27: Wound granulating rapidly. Temperature normal. Forearm fixed in different positions at daily dressing. January 12: Granulations flush with surface and much smaller. Can move elbow passively from 75° to 120° without pain. March 1: Can voluntarily flex from 150° to 90°. Remains under treatment.

DISCUSSION.

Mr. KENNETH GOADBY: I have been collaborating with Major Swan, and my remarks will be from the bacteriological standpoint. The lantern slides I exhibit show the high degree of septicity of such wounds. Besides ordinary pyogenic organisms, anaërobes (*Bacillus perfringens*, the bacillus of malignant œdema, *Bacillus Hibler IX*) are commonly present. The streptococci do not seem to be so virulent as those found in civilian practice, such as in post-mortem wounds, for the percentage of cases in which septicæmia results is very small. The anaërobic organisms are found in great numbers in the damaged bone tissue, especially in sequestra and in fragments of detached bone; this lends support to the method of treatment adopted by Major Swan, especially in view of the fact that organisms persist for many months in their comparatively secluded positions. I attach some importance to the preparatory course of mixed vaccines which has been employed. Since early treatment is desirable, I advocate the use of heterogeneous vaccines prepared from cultures grown on the blood of patients who have got well, and sensitized with the corresponding vaccines until an autogenous vaccine has been prepared.

The CHAIRMAN (Lieut.-Col. H. J. Waring): I agree in the main with the contentions of Major Swan. My experience has led me to prefer early excision and free drainage, with efficient flushing, to primary incision and excision at a later date. With regard to the muscular power which may follow excision of joints, I recall the case of a man whose elbow-joint was excised in pre-aseptic days, but who subsequently was able to carry on his work as a blacksmith.

Miss HELEN CHAMBERS: My observations, which have been made at the Women's Military Hospital, Endell Street, have shown that irrigation by solutions into septic wounds only renders the surface sterile when one of two solutions is used—eusol or Dakin's solution. Even then the result is only temporary. What is needed is some application the effect of which is continuous. Such can only be applied in the form of a paste. Bismuth-iodoform paste satisfies the condition and has proved very useful.

Major SWAN (in reply): I agree that excision of all the ends of bones is desirable provided that the gap produced is not unduly wide. I have not found any one solution of greatest value, but prefer a change from one solution to another. I am not convinced of the usefulness of bismuth-iodoform paste, as I have met with cases of failure in which I have had to remove it, and have

found the surrounding tissues in immediate proximity to the paste to contain many organisms. One of the great advantages of the early operation of excision is that free drainage of the septic area is provided for, whilst the mobility and functional use obtained in the joint fully compensate the patient for the inconvenience to which he is put. Excisions performed at a later stage do not yield such good functional results.

Section of Surgery.

SUB-SECTION OF PROCTOLOGY.

President—Mr. F. Swinford Edwards, F.R.C.S.

(*May* 9, 1917.)

Closure of Colostomy Opening for Carcinoma of Pelvic Colon by Greig Smith's Method.

By Percival P. Cole, F.R.C.S.

F. H. A., male, aged 65, was operated on in June, 1915, for carcinoma of the pelvic colon. The loop was exteriorized, the bowel and mesentery being removed by cautery a fortnight later. After treatment with rubber drainage tube after the manner described by Mitchell Banks, the colostomy was closed in September, 1915. The method adopted was that advocated by Greig Smith—extraperitoneal direct suture. The muscles of the abdominal wall were sewn together in two layers over the anastomosis. A small drainage tube was inserted and retained for forty-eight hours. There was no leakage and the parts healed without suppuration. The bowels were not opened till the fifth day.

The patient has never worn a belt, has never had any trouble ascribable to his operation, and has never experienced any feeling of weakness in his abdominal scar. It is noteworthy that his motions were evacuated normally for some weeks before closure was carried out.

The patient is shown to demonstrate the wide application of the extraperitoneal method, for his colostomy necessarily involved the whole circumference of the bowel and presented two separate orifices.

DISCUSSION.

Mr. Sampson Handley: I examined the patient myself, and it was a very good result; but, of course, the abdominal wall was necessarily rather thin over the seat of the operation. I take it that is inevitable with that method, but the result was very good, and there was no bulging.

The PRESIDENT: I should say it was a remarkably good case, because a large portion of the sigmoid colon had been completely removed, and therefore there must have been a tremendous spur formed, and we hear of no usage of an enterotome for destroying that spur. Of course, there are other ways of dealing with a spur, which so frequently forms after colostomy; and one means is by the insertion of a tube, such as we hear of in this case—namely, a Mitchell Banks' tube put in for some little time before. That seems to answer the purpose very well without the employment of an enterotome. But this must have been an aggravated case, so far as the spur was concerned, and I am surprised to learn that such a good result was obtained from the methods which were carried out.

(*May* 9, 1917.)

Case of Abdomino-perineal Excision in a Man, aged 29.

By J. P. LOCKHART MUMMERY, F.R.C.S.

A. S., AGED 29, was admitted to St. Mark's Hospital on June 10, 1916. There was a history of bleeding at stool and constipation for six months. Examination *per rectum* showed a fairly large carcinomatous growth occupying two-thirds of the lumen of the bowel just within the sphincters. A specimen which was removed for examination was reported to be adeno-carcinoma. A complete abdomino-perineal excision was performed with the establishment of a permanent colotomy, the operation being done under anoci-association anæsthesia. The growth was rather large and there was some difficulty in separating it from the bladder and prostate; a large portion of the prostate had to be removed with the growth. The patient made an excellent recovery and was up and about in three weeks. At the present time he can do a hard day's work, he has put on weight, and feels perfectly well. The colotomy, he says, causes him no serious inconvenience and does not interfere with his employment.

The PRESIDENT: This is a remarkably successful case, the more so as the patient is young, for in young cases a carcinoma grows rapidly. In addition, we have just heard that Mr. Mummery had to remove a portion of the prostate as well, showing that the growth was adherent to it, and was probably even involving it. That fact much increases the chance of recurrence. I trust, with him, that the patient may not have a recurrence for many years, if at all.

(*May* 9, 1917.)

Discussion on the Methods of making and closing Colostomy Openings.

Opened by J. P. LOCKHART MUMMERY, F.R.C.S.

THE earliest operations for making a permanent opening in the colon were extraperitoneal, the best known being Collinsen's or Amussat's lumbar colostomies. Littré's colostomy in the left groin was apparently not very successful at that time (1882), having according to Batt,[1] a mortality of 53 per cent.

With the advent of antiseptic surgery the necessity for an extraperitoneal route disappeared, and so-called inguinal transperitoneal colostomy became the fashion. From that time lumbar colostomy practically disappeared from surgical practice, and at the present time it must be a very unusual procedure. The left lumbar operation has, I think, still a possible field of usefulness in cases where a temporary colostomy is required previous to an abdominal section, for the advantages of the colon opening being well out of the way are obvious. It may also still be the safest operation in cases where there is obstruction and great distension of the colon with solid fæces. Personally I have never yet performed the lumbar operation except in the post-mortem room.

The inguinal operation as described by the Allinghams held the field until a few years ago. It was, I think, the Allinghams who first pointed out the importance of making a proper spur in performing the operation. Various modifications of the operation of inguinal colostomy have been suggested from time to time with the object of affording the patient greater control over the opening. Thus the operations described by Witzel, Braun,[2] Bailey and Weir,[3] all had for their object the formation of a valvular opening similar to Witzel's method of performing gastrostomy. I have tried these methods and have no hesitation in saying that they are not successful in attaining the object aimed at. In a very short time the valvular nature of the opening disappears, and the ultimate results of these operations compare unfavourably with the more usual operation, owing to the fact that a much larger wound in

[1] *Amer. Journ. Med. Sci.*, 1884, lxxxviii, p. 422.
[2] Braun, *vide* Bryant's "Operative Surgery," ii, p. 996.
[3] Weir, *Med. Record*, 1900, lvii, p. 666.

the abdominal wall is necessary, and a weak patulous opening is liable to result. The same remarks apply to methods such as Lilienthal's[1] operation, in which a twist is given to the bowel. Several other ingenious methods for obtaining control over the opening have been suggested from time to time, but I do not know of any that have stood the test of experience.

A very great improvement upon the old left inguinal colostomy was secured by making a vertical incision through the belly of the left rectus muscle. I first began to use this incision in 1907 and now never use the old oblique incision in the left iliac fossa, except for some very special reason which precludes the use of the rectus incision. It gives much better control over the opening, especially when the patient is standing or walking.

As regards giving a patient the best kind of colostomy opening, I consider the following factors in performing the operation to be of chief importance:—

(1) Bringing out the colon through as small an opening in the abdominal wall as possible.

(2) Making a good spur, and subsequently, completely dividing the bowel.

(3) Bringing the bowel through the rectus muscle.

(4) Providing a reservoir for the fæces immediately above the opening.

Unfortunately the last factor cannot be secured in the case of sigmoid colostomy without running the risk of a tiresome prolapse. It is applicable, however, to transverse colostomy.

During the last few years transverse colostomy has come a good deal into favour, and it has certain advantages. By making the opening in the ascending part of the transverse colon towards the splenic flexure an excellent natural reservoir is provided for the fæces immediately proximal to the opening. Another advantage is that prolapse of the bowel is very unlikely to occur. Personally I have not yet met with it. This is mainly due to the higher position of the opening in the abdominal wall. Prolapse is, after all, only another kind of hernia, and hernia above the umbilical level we know very rarely occurs. In most cases transverse colostomy gives excellent results as regards control, but I have met with at least two cases in which the stools have remained persistently loose after transverse colostomy, and this is certainly an objection, although it does not follow that a sigmoid colostomy would have been any better in these cases. It will be interesting to hear the experience of other surgeons on the advantages of transverse colostomy.

[1] Lilienthal, *Ann. of Surg.*, 1910, lii, p. 384.

Temporary Colostomy.

With regard to the best form of temporary colostomy, I do not think there is much to choose between transverse colostomy and sigmoid colostomy. Transverse colostomy is certainly preferable if the operation be performed as a preliminary to excision of the rectum, as the surgeon has the whole of the sigmoid to use in restoring the bowel and the opening is farther away from the site of operation. Apart from this, however, I think a sigmoid colostomy is the better for a temporary opening, but it is important that the centre of the sigmoid loop be used so that the colon can be readily mobilized when the opening has to be closed again.

Closure of a Colostomy Opening.

There is still considerable difference of opinion among surgeons as to the best method of closing a colostomy opening, and to judge from cases which I have seen at St. Mark's Hospital and elsewhere, quite a number of operations performed for this purpose are failures. Not infrequently I have cases sent to me in which more than one unsuccessful attempt has been made to close an opening in the colon. We may, I think, assume that in most cases a spur of some sort has originally been present.

I think it is obvious that the choice of method for closing such an opening must depend to some extent upon the ability of the operator and his previous experience of intestinal suture. A method which may be the best in the hands of an expert may be the worst in the hands of a comparatively inexperienced operator.

I think there is no doubt that the best method is to dissect the bowel out of the abdominal wall, free the loop sufficiently to allow the colon to be drawn well out of the abdomen, and then, after cutting away the edges of the opening, to restore the lumen by direct suture. The colon is then replaced in the abdominal cavity and the wound in the abdominal wall sewn up. I attach considerable importance to the following points in performing the operation:—

(1) Before commencing the operation the external mucosa is either thoroughly sterilized, or is dissected loose and the opening temporarily closed with suture.

(2) An incision having been made around the opening, it is deepened on one side until the abdominal cavity is opened; then, with one finger inside the abdomen as a guide, the bowel is freed from the abdominal wall with scissors.

(3) The loop is drawn through a hole in a towel and, after being clamped on the proximal side, a wedge-shaped piece of the bowel with its apex towards the mesenteric attachment is cut away (the mesenteric side of the bowel can as a rule be left) so as to secure a good blood supply to the anastomosed edges, and to compensate for the narrowed lumen at the site of the join.

(4) The ends are sewn together with a catgut stitch taking up all the coats, and a serous stitch, also of catgut, is placed over this.

(5) The bowel is cleaned carefully and, the gloves and instruments having been changed, is replaced in the abdomen.

(6) The abdominal wall is closed with a small rubber tissue drain at the lower corner of the wound.

(7) After operation the bowels are not confined but are kept acting daily by small enemata or small doses of magnesium sulphate by the mouth.

The advantages of this method are, I think, obvious, but good technique is essential.

The oldest method, and certainly the easiest, is by division of the spur with an enterotome, and subsequent closure of the external fistula by paring away the edges, inverting them, and bringing together the abdominal wall with deep sutures. This method appears to be very safe and the results are very good. Provided the spur is completely divided the subsequent closure of the opening is not difficult. There is no fear of stricture at the site of union, and the risk of the operation appears to be very small. Although the operation must have been done a great number of times I have not been able to find any recorded fatalities from it, though, of course, this does not mean that none have occurred. I have closed colostomies a number of times by this method without any failures, and if the patient is a bad risk, and safety is the only consideration, I think it is probably the best method.

Greig Smith's[1] operation, in which the bowel is sutured extraperitoneally, does not seem to me to have any advantages over either of the previous methods, and to belong to the time when surgeons were afraid to open the peritoneal cavity and preferred lumbar to inguinal colostomy. I have performed it several times in the past with quite good results as regards closure of the colostomy, but it leaves the gut outside the peritoneum and with a narrowed lumen. The patients generally have chronic constipation, and a weak place in the abdominal wall which requires the use of a support, and I shall certainly not use it in the future.

[1] "Abdominal Surgery," 5th ed., 1897, p. 729.

Coffey's[1] operation, which is somewhat similar, is open to the same objections.

With regard to the young men wounded in the present war for whom it is necessary to close a temporary colostomy, it seems to me most desirable that we should, as far as possible, use that method which will give perfect anatomical restoration of the parts.

The question of the amount of incapacity that is caused by the establishment of a permanent colostomy opening is one that has not, I think, received much attention, although it is of considerable importance, and should not be very difficult to settle. In order to ascertain as far as possible what the real facts are with regard to the incapacity caused by a permanent colostomy, I have taken fifty cases of colostomy from my own case books and classified them according to the amount of control over the opening that they possessed at the end of three months from the date of the operation. They were classified as follows:—

(1) *Excellent* control when the patient could live an ordinary life without the fear of an accident occurring.

(2) *Good* control when the patient could live an ordinary life but there were occasional accidents.

(3) *Poor* control when the patient had to be very careful and accidents were not infrequent.

(4) *Bad* when there was no control over the stools.

Out of the fifty cases there were seventeen cases of excellent control. Of these fifteen had either had the growth removed, or the colostomy had been done for a non-cancerous condition. In only two of these cases had the operation been done for inoperable cancer. The average age of the excellent cases was 49·1. In four of these cases transverse colostomy was done.

Twenty-three cases had good control. Thirteen of these were cases of inoperable cancer. The average age of the good cases was 55·5.

In six cases the control was poor. Five of these were cases of inoperable cancer. The average age was 67·1. Two were transverse colostomies.

In four cases the control was bad. Of these two were inoperable cancer cases, and two cases in which the growth had been removed. The average age was 59·75. One case was a transverse colostomy. Two of the patients were over 70 and bedridden.

[1] *Ann. of Surg.*, June, 1907.

The results given in percentages were as follows: Excellent, 34 per cent.; good, 46 per cent.; poor, 12 per cent.; bad, 8 per cent.

This seems to me to prove what one would have expected—namely, that when the patient is in good health with no growth, and not old, the

No.	Age	Sex	Cause of operation	Kind of colostomy	Control after three months
1	50	M.	C. R.; inoperable	Sigmoid	Excellent
2	62	M.	C. R.; removed	Sigmoid	Good
3	45	M.	C. R.; inoperable	Sigmoid	Good
4	60	M.	C. R.; removed	Sigmoid	Good
5	60	F.	C. R.; removed	Sigmoid	Good
6	58	M.	C. R.; inoperable	Sigmoid	Good
7	53	M.	C. R.; removed	Sigmoid	Excellent
8	75	M.	C. R.; inoperable	Sigmoid	Poor
9	68	M.	C. R.; removed	Sigmoid	Excellent
10	60	M.	C. R.; removed	Sigmoid	Excellent
11	64	M.	C. R.; inoperable	Sigmoid	Good
12	50	M.	C. R.; removed (A. P.)	Sigmoid	Excellent
13	67	M.	C. R.; inoperable	Sigmoid	Poor
14	35	F.	C. R.; unoperated	Sigmoid	Good
15	62	M.	C. R.; inoperable	Sigmoid	Good
16	49	F.	C. R.; removed (A. P.)	Sigmoid	Bad
17	51	M.	C. R.; inoperable	Sigmoid	Good
18	49	M.	C. R.; removed (A. P.)	Sigmoid	Good
19	37	M.	C. R.; removed	Sigmoid	Excellent
20	56	M.	C. R.; inoperable	Sigmoid	Good
21	50	M.	C. R.; inoperable	Sigmoid	Excellent
22	62	M.	C. R.; inoperable	Sigmoid	Good
23	72	M.	C. R.; inoperable	Sigmoid	Bad
24	42	M.	C. R.; inoperable	Sigmoid	Good
25	54	M.	C. R.; inoperable	Sigmoid	Good
26	42	M.	Pericolitis	Sigmoid	Good
27	50	M.	C. R.; removed (A. P.)	Sigmoid	Excellent
28	55	F.	C. R.; removed	Sigmoid	Good
29	57	M.	C. C.; removed	Sigmoid	Good
30	75	F.	C. R.; removed	Transverse	Poor
31	63	M.	C. R.; removed	Sigmoid	Good
32	71	F.	C. R.; inoperable	Sigmoid	Poor
33	48	M.	C. R.; inoperable	Transverse	Poor
34	60	M.	C. C.; removed	Sigmoid	Excellent
35	52	M.	C. R.; removed	Transverse	Excellent
36	60	M.	Pericolitis	Transverse	Excellent
37	62	F.	C. R.; inoperable	Sigmoid	Good
38	67	M.	C. R.; inoperable	Sigmoid	Poor
39	53	M.	C. R.; removed	Transverse	Good
40	52	M.	C. R.; removed	Transverse	Excellent
41	72	F.	C. R.; inoperable	Transverse	Bad
42	24	F.	Stricture	Sigmoid	Excellent
43	45	M.	Pericolitis; removed	Sigmoid	Excellent
44	42	F.	C. R.; removed	Transverse	Excellent
45	29	M.	C. R.; removed (A. P.)	Sigmoid	Excellent
46	72	F.	C. R.; inoperable	Sigmoid	Good
47	46	M.	C. R.; removed	Sigmoid	Bad (became good later)
48	54	M.	C. R.; removed	Sigmoid	Excellent
49	60	M.	C. R.; removed	Sigmoid	Good
50	52	M.	C. R.; removed	Sigmoid	Good

colostomy does not cause any serious inconvenience in the vast majority of cases; and that with very few exceptions, the cases in which a colostomy opening causes a patient serious inconvenience are those in which an inoperable growth exists and the patient is old and feeble.

In my experience patients who have had a colostomy opening over five years usually suffer extraordinarily little inconvenience from it and are quite satisfied with their condition. I estimate that a permanent colostomy opening in a patient not suffering from cancer (or in whom the growth has been successfully removed), and not over 60 years of age at the time of operation, ranks as regards the amount of incapacity it causes as decidedly less than the loss of one foot by Syme's amputation.

I would suggest that this Sub-section should appoint a committee to collect a large number of statistics on this question of incapacity and carefully analyse them for the benefit of the profession.

Mr. SAMPSON HANDLEY: It seems to me that Mr. Mummery is right in insisting on the value of the rectus sheath incision. I have used no other method during the last few years, and I think the improvement over the old inguinal colostomy is enormous. The control is generally very good, and the only trouble I have seen—and that in only a single case—was stricture, which was satisfactorily dealt with by a secondary operation. The sigmoid is not a very suitable bowel to employ when one uses the rectus sheath opening. I think it means bringing it across too much. For that reason alone, the transverse colon should, I think, be chosen. But perhaps the most important reason for choosing the transverse colon is the advantage it gives in stout patients, for in them inguinal colostomy may be a most difficult operation, and it may be impossible to bring the sigmoid up to the level of the abdominal wall if there is great distension present, or if the sigmoid is bound down, even when the outer layer of the sigmoid colon has been divided. Whereas the transverse colon, even in the stoutest patients, is always freely movable, and can be brought to the level of the skin. Furthermore, the transverse colon is farther away from the region of the growth, a point which may be of great importance in relation to those growths which advance along the length of the bowel and involve a considerable number of inches of the colon. There is a technical point to which I would like to draw attention, as I have not seen any reference to it in print. When the transverse colon is brought down through the abdominal wall, a clumsy mass of fat protrudes with it, and it is embarrassing to the operator to have to deal with it. That can be avoided very simply by scratching through these layers of the omentum in front of the transverse colon, and insinuating the instrument round, so that there is only one layer of peritoneum brought out; the remainder is behind the glass rod and inside the peritoneal cavity. I have found that that method simplifies and facilitates the

operation very much. With regard to the discussion as between the extra- and the intraperitoneal methods of closing a colostomy opening, I have no very strong views. I have not had enough experience of the intraperitoneal method to enable me to say it is superior to the extraperitoneal. But it appeals to me as a logical and thorough method, on the lines of sound modern surgery, and I see no reason why it should be more dangerous than the other, especially if Mr. Lockhart Mummery's valuable suggestion of keeping the bowels loose after the operation is adopted.

Mr. ASLETT BALDWIN: It always seems to me that if a person has got a colostomy which should be closed, it is a pity to subject him to more risks than are absolutely necessary. The method I have always used for closing colostomies has been a modification of Greig Smith's method of extraperitoneal closure. I make an incision through the skin, about $\frac{1}{16}$ in. from the opening, all round, go through the whole thickness of the skin, and separate the bowel with this piece of skin attached. I sew up the opening with chromicized catgut, leaving the ridge of skin all round, and including it and the bowel in the suture: the skin prevents the suture from cutting out easily. This suture line is then inverted with a second chromic catgut suture. The bowel, by means of the finger, is separated all round, and is pushed deep to the whole of the abdominal muscles. Then the internal oblique and the transversalis are brought together with catgut over the bowel, and the external oblique is brought over that, and, finally, skin. A small drainage tube is put down in the centre, near the bowel but not quite close to it. That is taken away on the second or third day after the bowel has been moved. Under that treatment I see no reason for danger; the operation is perfectly easy, and there is no reason why there should be any permanent weakening of the abdominal wall, though it has been suggested that there might be. I think, Sir, you expressed surprise that there was no more weakening in the case which was shown just now. I do not see why these should have weakening if the muscles are brought together again. With regard to narrowing of the lumen of the bowel, I think that is negligible. If the bowel is narrowed, I think it dilates up later and compensates for the slight narrowing, and I do not remember such a case in which the patient has had inconvenience on account of constipation. It seems to me that is the ideal method of closing colostomy openings. It is, I contend, free from all risks, and it is easy to do. The spur disappears as the bowel is pushed back. The ridge of skin causes no trouble, its epithelium no doubt adapts itself to its new environment, if any is left, but I cut away the epithelial line with curved scissors before suturing.

The PRESIDENT: Mr. Baldwin says he has not met with interference with the lumen of the bowel owing to spur formation, but this must depend on what was done at the time of the operation—i.e., whether any part of the circumference of the bowel was removed. Some surgeons cut off a good deal, while others merely incise it.

Mr. BALDWIN (answering the President): Where there is a chance of closing a colostomy no bowel would be cut away. It would be incised, probably longitudinally.

The PRESIDENT: For many years past I have taken much interest in colostomy, colotomy and closure of colostomy openings, and fifteen years ago I adopted hypogastric colostomy, as opposed to iliac. This was acting on a hint which I got from the late Mr. Bidwell. He pointed out that control was likely to be much better if the bowel was brought up through the rectus muscle: so in most cases I have done colostomy through the left rectus muscle, and I have found that, in many cases, control has been excellent. I have had people who were able to play as good a game of golf after as before this operation. I recollect the case of a lady who, for fifteen years, had a colostomy, and used to go into society, to the theatre and to dinners, without being a nuisance to her companions. I prefer this to transverse colotomy, in which one has to deal with the omentum which lies in front of the transverse colon. Sometimes it is very thickened, and one has, as Mr. Sampson Handley said, to divide it to bring up the transverse colon. The omentum is more marked in some cases than it is in others; sometimes one hardly notices it at all. An accident happened to me once in doing a transverse colostomy. The opening having been made in the abdominal parietes, I defined the transverse colon, and after scratching through the omentum passed my finger and thumb around the gut, as I wanted to feel the posterior wall of the bowel, so as to act as a guide for the insertion of a deep suture. But in spite of this I found I had perforated a piece of small intestine. Fortunately it was seen in time, and I was able to close the opening in the small intestine with no untoward result. I have not had that happen to me during either inguinal or hypogastric colotomy. As to closure, I have almost invariably made use of the extra-peritoneal method. And in reference to that, I am indebted to another colleague of mine, Mr. Baldwin, who has just given us the benefit of his opinion on this subject. It was he who directed my attention to the fact that it would be well to leave a small margin of skin all round the mucous membrane, in order that the sutures should hold better than when passed through mucous membrane alone. I do not say one always gets success at the first operation; indeed, I have had to operate three times in order to get perfect closure. But I have never lost a case, and I have operated upon a good many. I have derived a valuable hint from Mr. Mummery's paper, and that is with regard to the intraperitoneal method of closing a colotomy—viz., after isolating the artificial anus, to close it by suturing the mucous membrane before opening the peritoneum. If that practice were carried out, I think it would limit, to a great extent, the risks of the intraperitoneal operation. It is a more ideal operation, to my mind, because the bowel can be quite replaced in the belly, which is not always possible by the extraperitoneal method. I shall hope to have the opportunity of trying that method some day. With the rest of the paper I thoroughly agree, except that I do not see the advantage of transverse colotomy over hypogastric, except where the growth is too high-

lying to permit of the colon being opened above it through the hypogastric incision, also in cases where it is advisable to have the artificial anus as far removed as possible from any future operation area.

Mr. ASLETT BALDWIN: I do not know, Sir, whether you remember a case some years ago in which I was helping you to do a colostomy in a very stout patient, where the difficulty mentioned by Mr. Handley was present. It was a left inguinal colostomy, and the bowel could not be got up. The belly wall had many inches of fat, and I suggested to you that you should cut the fat away, in that way bringing the skin down to the colon. You did that, and the operation was managed perfectly well. It would have been impossible without cutting away sheets of fat.

(The PRESIDENT: Yes, I remember it.)

Mr. J. P. LOCKHART MUMMERY (in reply): I am interested to note that there seems to be general agreement with regard to the value of the rectus incision in colotomy. I agree with Mr. Handley that the proper method of doing a transverse colotomy is to scratch a hole in the omentum and bring a knuckle of bowel through it, instead of puckering up the omentum round the wound. I have always used this method. With regard to transverse colostomy, I am still open to conviction as to whether this is the best form of colostomy. There are certain cases in which transverse colostomy offers obvious advantages. I have had one or two cases in which I could not do transverse colostomy, because the transverse colon was very short, and one could see it would cause a drag on the lower border of the stomach, which is obviously not advisable. I think making an opening fairly high up is of value, because of the reservoir produced in the bowel immediately behind the opening. Mr. Handley raised the question of sigmoid colostomy through the rectus. I have had no trouble in doing transverse colostomy through the rectus: I have dragged the sigmoid up, and I have had no difficulty in getting it to come through the rectus incision. If there seems to be any difficulty, I pull down the nearest piece of transverse colon. I make an incision just below the umbilicus, and one can then do a sigmoid or a transverse colostomy, whichever seems preferable. With regard to the question of the extraperitoneal method of closing a colostomy, most of those here are in favour of the Greig Smith operation, but I think that, as time goes on, they will come round to the intraperitoneal operation. In Mr. Baldwin's procedure I do not like the leaving of skin inside the bowel. Practically it may be all right, but theoretically one would think it might be dangerous, as one does not know how skin would behave in such circumstances. Most of the closures of colostomies have been done on elderly people, who may not have many years of life before them, but we have now to face the large number of wounded from the Front, and to realize that closure of colostomies must be done on young boys; and certainly in those cases we must do the operation which will, as far as we can secure it, leave a perfect anatomical condition.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

J. Y. W. MacALISTER

UNDER THE DIRECTION OF

THE EDITORIAL COMMITTEE

VOLUME THE TENTH

SESSION 1916-17

SECTION OF THERAPEUTICS & PHARMACOLOGY

LONDON
LONGMANS, GREEN & CO., PATERNOSTER ROW
1917

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SECTION OF THERAPEUTICS AND PHARMACOLOGY.

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LONDON:
JOHN BALE, SONS AND DANIELSSON, LTD.,
OXFORD HOUSE,
83-91, GREAT TITCHFIELD STREET, OXFORD STREET, W. 1.

Section of Therapeutics and Pharmacology.

President—Dr. W. Hale White.

(*February* 20, 1917.)

The Principles of the Transfusion of Blood.

By A. E. Stansfeld, M.D., M.R.C.P.

The idea of transfusing blood from the healthy to the sick has appealed to the imagination of our profession for centuries, but no considerable progress was made until recent years, owing on the one hand to the difficulties which the operation presented, and on the other hand to the dangerous reactions which sometimes followed the more successful attempts. But the revived enthusiasm for transfusion during the last decade in America, France and Germany has finally established its value upon a sound experimental and clinical basis, and it is remarkable that the subject has received so little attention in this country. The whole of Europe has been preoccupied, however, during the last three years, and I may remind you, therefore, that transfusion has been successfully employed in the treatment of various kinds of anæmia and in the arrest of spontaneous hæmorrhage, whilst in combating serious infections and certain toxæmias, such as the toxæmias of pregnancy, promising results have been obtained.

How far the important factors of the blood concerned in achieving these various results reside in the serum it is not yet possible to say. So far as disintoxication and immunity are concerned transfusion may be merely a convenient method of transferring healthy or immune serum to the patient, and with the manifest domain of serum therapy I do

not propose to deal. But blood therapy is still in its infancy; it could not be properly tested until an elaborate technique had been perfected and certain accidents liable to result from the chance admixture of different bloods had been excluded, and so, with the remedy in our hands, we have yet to learn exactly when and in what doses to use it.

Selection of Cases.

The most obvious application of transfusion is to the treatment of anæmia, but there are as yet very few guides to the selection of suitable cases. The advantage of transfusion in the case of patients who have been rendered anæmic by recent hæmorrhage is sufficiently clear, but it is probable that the average existence of the transfused cells in the circulation of the recipient is not longer than ten days, even under the most favourable conditions, and therefore in cases of anæmia due to other causes, the added blood, although it may serve to tide over a crisis, can do no permanent good unless blood formation is stimulated or blood destruction diminished in the patient's own body.

That an increased activity of the bone-marrow frequently follows transfusion is demonstrated by the fact that the blood count usually rises with a simultaneous increase in the number of reticulated red cells. But when the condition of the peripheral blood indicates a hypoplasia of the bone-marrow, the only method of determining whether increased blood formation will result from transfusion is to transfuse and see. The following case is probably an example of simple stimulation of the bone-marrow:—

Case I.—J. L., female, aged 11, was admitted to St. Bartholomew's Hospital under the care of Dr. Morley Fletcher, suffering from a severe anæmia which had followed an attack of diphtheria eight months previously. No other cause than a simple hypoplasia of the bone-marrow could be found to account for the anæmia, and as a result of the first six weeks' treatment the red cell count rose from 925,000 per cubic millimetre to 1,330,000 per cubic millimetre, the hæmoglobin from 18 to 31 per cent. The patient then received 20 c.c. of blood from the House Physician, and in twelve days the blood showed 1,580,000 red cells per cubic millimetre and 45 per cent. hæmoglobin; 150 c.c. of blood was then transfused, and during the next seventeen days the red cells rose to 3,000,000 per cubic millimetre and the hæmoglobin to 63 per cent. Thereafter the anæmia diminished more slowly until the patient went to a convalescent home.

In those diseases, such as pernicious anæmia, in which an excessive hæmolysis is known to occur, it is difficult to determine how far improvement following transfusion is due to increased cell production, and how far it may be due to diminished hæmolysis. That the latter is one of the factors concerned might be expected on clinical grounds, for, as with spontaneous remissions, the patient becomes less yellow and toxic in appearance, fever diminishes, and a feeling of well-being which is out of proportion to the change in the condition of the blood is soon experienced. The observations of Wilbur and Addis, Robertson and others, have shown that the variations in urobilin output in cases of pernicious anæmia not only serve as an index of blood destruction but correspond to the remissions and relapses in the course of the disease, whether these be spontaneous or attributable to treatment; and Robertson has shown that the remission following transfusion in a case of pernicious anæmia was accompanied by a corresponding diminution of urobilin excretion.

In some cases of pernicious anæmia the myeloid reaction becomes less evident as the patient improves after transfusion, again suggesting that marrow hyperplasia is not wholly responsible for the improvement. This is illustrated by the following case:—

Case II.—Mrs. H., aged 42, suffering from pernicious anæmia with an enlarged spleen, was admitted to St. Bartholomew's Hospital under the care of Dr. Drysdale. Frequent examination of the blood suggested great hæmopoietic activity, 4,000 to 6,000 normoblasts and 100 to 400 megaloblasts per cubic millimetre being constantly present, but in spite of all treatment the anæmia steadily increased, until, three months after admission, the blood showed 429,000 red cells per cubic millimetre and 19 per cent. hæmoglobin. At this stage the patient, who had developed bronchitis, appeared to be moribund, but after receiving 250 c.c. of blood from her husband she showed a rapid and remarkable clinical improvement. During the next five weeks the blood count rose to 2,550,000 per cubic millimetre and the hæmoglobin to 66 per cent., while the nucleated red cells as steadily disappeared from the peripheral blood. The patient was then transfused again and improved still further, the red cells reaching 3,850,000 per cubic millimetre and the hæmoglobin 83 per cent. There were signs of relapse before she left the hospital, at her own request, thirteen months ago, but it is now more than two years since the onset of the disease, and she is still able to look after her own home.

The ultimate prognosis in any case must depend upon the power of reaction in the bone-marrow, and this, so far as I know, is only to be determined by trial.

I have transfused nine cases of pernicious anæmia, eight of whom were selected because they were making no progress or steadily becoming worse under other methods of treatment, the ninth being chosen because her condition was regarded as too serious to justify a trial of other methods. Of these nine, four showed very marked improvement, three, of whom two are still under treatment, showed definite but less marked improvement, one was merely kept alive for several weeks, and one derived practically no benefit. So far as these cases were concerned, the age of the patient, the duration of the disease, and the condition of the bone-marrow as indicated in the peripheral blood proved to be the best guides to the progress subsequent to transfusion; but in some cases the results far exceeded expectation and in others they were disappointing.

Selection of Donors.

The donor should be a healthy adult, and the loss of such amounts as I have generally used (250 c.c.) is never felt. In America large quantities, even up to 1,800 c.c., have been taken, and although such loss must debilitate the donor, there is probably no objection to taking as much as 500 c.c. at one time.

McClure has recently recorded the transmission of syphilis from donor to patient. The donor, who had denied all possibility of infection, had a primary sore at the time of operation, and six weeks later both the donor and the patient, who was suffering from pernicious anæmia, simultaneously developed secondary eruptions. It seems unlikely that syphilis would be transmitted by a donor, who, on careful examination, showed no signs of disease, but it is desirable that a Wassermann reaction should be done on the blood if time and facilities permit. In most cases, however, the donor is a relative or friend of the patient, and he may be expected to answer questions frankly if the danger involved is explained to him.

McClure also reports the transmission of malaria from a donor in whom the disease was quiescent to a patient in whom it flared up. But these two cases are the only instances I can find of the transmission of disease.

If the prospective donor is found to be healthy his blood should be tested with that of the patient for isoagglutinins and isohæmolysins. Landois, in 1875, was the first to recognize the incompatibility of certain bloods, and Landsteiner, in 1901, divided individuals into three

groups, according to the isoagglutinative properties of their bloods. In 1910 Moss recognized a fourth group, and the accompanying chart shows the reactions of the various groups (Chart I).

CHART I.

Group	Serum agglutinates	Corpuscles agglutinated by
I	*Nil*	II, III, and IV
II	I and III	III and IV
III	I and II	II and IV
IV	I, II, and III	*Nil*

CHART II.

		CORPUSCLES											
		T. B.	A. E. S.	A. R. D.	B.	L.	N. F.	V.	A.	E. G. D.	H. S.	T.	C. S.
SERUM	T. B. ...	...	—	—	—	—	—	—	—	—	—	—	—
	A. E. S.	a	...	—	—	—	A	—	—	—	—	—	—
	A. R. D.	A	—	...	—	—	AA	—	—	—	—	—	—
	B. ...	A	—	—	...	a	AA	—	—	—	—	—	—
	L. ...	AA	—	—	—	...	AA	—	—	—	—	—	—
	N. F. ...	AA	AAA	AA	AA	AA	...	—	—	—	—	—	—
	V. ...	AA	AAAA	AAA	AAA	AAA	AA	...	—	—	—	—	—
	A. ...	AAA	AAA	AAA	AA	AAA	AAA	—	...	—	—	—	—
	E. G. D.	AAA	AAAA	AAA	AAA	AAA	AAA	—	—	...	—	—	—
	H. S. ...	AAA	AAA	AAA	AAA	AAA	AAA	—	—	—	...	—	—
	T. ...	AAAA	AAAA	AAAA	AAAA	AAAA	AAA	—	—	—	—	...	—
	C. S. ...	AAAA	AAAA	AAAA	AAAA	AAAA	AAA	—	—	—	—	—	...

a = very slight but definite agglutination.
A = well-marked agglutination.
AAAA = complete agglutination.

Chart II shows the result of testing together twelve specimens of blood. The first person belongs to Group I and possesses no agglutinins, but his corpuscles are agglutinated by the serum of all persons in other

groups. The next four persons belong to Group II, the sixth to Group III, and the last six to Group IV. It should be noted that the fourth person (B.) slightly agglutinates the corpuscles of someone belonging to the same group, and it may be noted, too, that two of the specimens belonging to different groups, one (A. R. D.) in Group II and one (E. G. D.) in Group IV are from brothers. The technique employed in making these tests was that described by Dudgeon and Wilson.

These four groups described have been supposed to be sharply defined so that all bloods falling into one group are mutually compatible, while they are incompatible with all bloods in the other groups. It will be seen that if this were true the group to which any blood belonged could be fixed by testing it against a known blood belonging to either Group II or Group III. My own observations, however, show that a certain amount of overlapping of the groups occurs, and this is well illustrated by Chart III, in which it will be seen that Mrs. H. belongs to Group II, so that her serum agglutinates the corpuscles of the persons in Group III but has no influence upon those of her own group. Mr. H.'s corpuscles, then, should be unaffected by the sera of Group III, but it was found that two of the Group II sera produced slight agglutination, while of the Group III sera one failed altogether to agglutinate, and the other two acted no more strongly than the sera of Group II.

It is generally admitted, moreover, that different bloods contain different amounts, as well as different kinds, of agglutinin, and that the amount of agglutinin in a single blood may vary slightly from time to time. That these variations may be sufficient to obscure the true grouping of a particular blood is illustrated by the case of the patient C. S., who was transfused from the donor N. H. (*see* Chart III).

Case III.—On November 1, 1915, C. S., a patient suffering from pernicious anæmia under the care of Dr. Feiling in the Metropolitan Hospital, received 250 c.c of blood from N. H., after the usual tests had shown the absence of agglutinins and hæmolysins. The red cells were immediately raised from 625,000 to 810,000, but a week later they had fallen to 600,000, and the patient appeared to have derived no benefit from the operation. There was, however, no unfavourable reaction of any kind. On November 22 the operation was repeated. When the patient had received about 20 c.c. of blood he complained of feeling faint, and became even more pallid. As the transfusion continued his pulse became very feeble, he retched and vomited violently, his face became distinctly puffy, and his skin was covered with cold sweat. When he had received about 100 c.c. he complained of pain in the sacral region, and this quickly became so intense that he could only be quieted with morphia;

250 c.c. of blood were given, and a few hours afterwards the patient began to pass hæmoglobin in the urine. Twelve hours later he was a deep yellow colour, and slightly œdematous all over, but he said that he felt much better. The hæmoglobinuria ceased after thirty-six hours, the yellow colour and the œdema disappeared in the course of about three days, and the patient's general condition steadily improved until six weeks later, when he left the hospital for domestic reasons, he had just over 3,000,000 red cells per cubic millimetre and 75 per cent. of hæmoglobin. (It is interesting to note the improvement which followed transfusion in this case in spite of immediate hæmolysis of the transfused blood.)

Subsequent examination of the blood revealed the fact that C. S. belonged to Group IV and N. H. to Group III, but apparently N. H.'s corpuscles were not very susceptible to agglutination, and the titre of C. S.'s serum at the time of the first transfusion must have been so low as to give no agglutination by the ordinary tests. It appears, therefore, that a single examination is not always sufficient to establish the compatibility of two bloods on all future occasions.

CHART III.

SERUM	CORPUSCLES								
	A. E. S.	Mr. H.	A. R. D.	R.	Mrs. H.	N. F.	L. F.	N. H.	C. S.
A. E. S.	...	—	—	—	—	a	a	—	—
Mr. H.	—	...	—	—	a	a	A	—	—
A. R. D.	—	—	...	—	—	AA	AA	a	—
R. ...	—	—	—	...	a	AAA	A	a	—
Mrs. H.	—	—	—	—	...	AAAA	AAAA	A	—
N. F....	AAA	AAA	AAA	AAA	a	...	—	—	—
L. F. ...	AAA	AAA	AA	AA	—	—	...	—	—
N. H....	AAA	a	AAA	A	a	—	—	...	—
C. S. ...	AAA	AA	AAA	AAA	A	A	AA	AA	...

a = very slight but definite agglutination.
A = well-marked agglutination.
AAAA = complete agglutination.

There is no evident familial grouping of bloods. I have already remarked that two of the bloods in different groups in Chart II belong to brothers and I have observed such differences in several other cases, but, unfortunately, opportunity has not permitted a further study for determining whether similarity or dissimilarity of blood reactions accompanies other physical features.

The group to which the blood belongs does not appear to be determinable at birth. Cherry and Langrock recently examined the blood of thirty-four infants, each against the blood of its own mother, without finding a single instance of agglutination. Hektoen states that the reaction of the child's blood is evident by the sixth year, and other observers have found the grouping to be apparent even before this, but it is useful to know that an infant may be safely transfused from its mother without performance of the usual tests.

Hæmolysins are found less frequently than agglutinins and it is established that they never occur without agglutinins. Ottenberg, Kaliski and Friedman, experimenting on dogs, found that hæmolysins were sometimes developed in the serum after repeated transfusions of agglutinable cells, but they were never developed by the transfusion of non-agglutinable cells. It is possible that the patient, C. S. (Case III), developed a hæmolysin, or more hæmolysin and more agglutinin, as a result of the first transfusion.

It has been noted by several observers that, as in Case III, hæmolysis following transfusion may be a much more violent phenomenon than hæmolysis of the same blood *in vitro*, and even a slight degree of agglutination of the proposed donor's corpuscles should be viewed with suspicion, for the danger is not one to be lightly regarded. Bernheim, collecting 800 reported cases of transfusion, found fifteen instances of macroscopic hæmolysis. Among these there were four deaths, three in cases in which no preliminary tests had been made and one in which it had been found that the donor's cells were slightly agglutinated by the patient's serum. There can be no doubt that a much higher proportion of disasters would have occurred but for the safeguard of preliminary tests.

Moss showed that antihæmolysins are always present in blood the red cells of which are hæmolysed by the serum of another individual. The presence of the antihæmolysin and the dilution of the transfused blood by the blood of the recipient probably explain the fact that it is less dangerous to introduce a blood containing hæmolysins for the

patient's corpuscles than to introduce one of which the red cells are destroyed by the patient's serum.

I know of no immediate phenomena resulting from the introduction of cells which are agglutinated without being hæmolysed by the recipient, nor indeed is it known whether isoagglutination occurs in the circulation. On one occasion, in the absence of a more suitable donor, I gave a patient 100 c.c. of blood the red cells of which he agglutinated. He was not benefited, but on the other hand he exhibited no unfavourable reaction. Nevertheless, it is undesirable to run risks by employing a donor belonging to a different group to the patient except in cases of extreme urgency and then the risk will be least if the donor belongs to Group IV, so that his own cells cannot be agglutinated or hæmolysed. Or if in such cases the grouping of the bloods of both donor and recipient is unknown and testing is impracticable, a small preliminary transfusion should be done half an hour before the main mass of blood is transfused, so that gross incompatibility may be recognized in time.

Reactions following Transfusions of "Compatible" Blood.

Even in those cases in which both donor and recipient have been found to belong to the same group, febrile reactions are apt to follow transfusion. Among my own cases rigors have occurred in about 10 per cent., fever in about 25 per cent., and these figures agree closely with those published by most observers. The rigors have followed within half an hour of the transfusion and the temperature in the febrile reactions, apart from the sudden rise accompanying the rigor, has usually reached a maximum of 102° F. to 103° F. on the following day, subsiding within forty-eight hours.

It has been suggested that the fever is due to incipient coagulative changes in the transfused blood and some support is lent to this view by the fact that rigors are common after intravenous injection of defibrinated blood. Moss noted rigors and transient fever in the majority of seventy-five cases which he treated by this method without other untoward results. But I have noticed a minute trace of coagulum remaining in my apparatus after transfusion on several occasions although no reaction followed in the patient; and Lindeman, in a series of 108 transfusions, observed that febrile reactions, with or without rigors, occurred much less commonly when the donor was a relative of

the patient than when he was an "alien," and this although incompatibility due to agglutination or hæmolysis has been ruled out in every case.

In some of my cases in which the patient has been transfused twice from the same donor, a febrile reaction has followed the first transfusion but not the second; in other cases no reaction followed on the first occasion, while the second was succeeded by fever. But only in one case have the symptoms been at all alarming.

Case IV.—A. C., aged 38, suffering from pernicious anæmia, was admitted to St. Bartholomew's Hospital under the care of Dr. Morley Fletcher. During a previous admission I had twice transfused him from his brother with marked benefit, the red cells rising from 500,000 to 3,800,000 per cubic millimetre, and the hæmoglobin from 16 to 86 per cent. during the course of seven weeks. He was now transfused with 275 c.c. from another donor who had made a long journey for the purpose and whose blood had been found to be compatible a week previously. Within fifteen minutes of the completion of the operation the patient had a rigor and his temperature rose steadily until it reached 105° F. three hours later. He became delirious and on the day following the operation the yellow colour of the skin was much deeper than before, but the blood serum showed no trace of hæmoglobin. The most alarming symptom, however, was complete anuria, which lasted for forty-three hours after the transfusion. The urine passed subsequently contained an excessive amount of urobilin but not an unusual amount for a case of pernicious anæmia. The temperature fell slowly, reaching normal three days after the transfusion. But the subsequent rise in the number of red cells and the amount of hæmoglobin was slight and transient and the patient is about to be transfused again. Unfortunately, owing to the remoteness of the donor, I have been unable to repeat the agglutination tests in this case.

There are, therefore, certain reactive phenomenon which we cannot at present avoid, although the frequency of their occurrence is said to be diminished by the choice of close relatives of the patient as donors. The chief dangers may be eliminated by careful testing for agglutinins and hæmolysins, but it may prove desirable to investigate other factors of which we as yet know nothing, not merely for the sake of avoiding accidents but also to determine whether a given donor is likely to afford the maximum benefit in a particular case.

Dosage.

The amount of blood to be transfused depends upon the purpose of the transfusion, and in cases in which a considerable transfusion is proposed without the immediate object of increasing the red cell content of the blood it may be advisable to bleed the patient before the operation in order to avoid overloading the circulation.

For the arrest of spontaneous hæmorrhage small doses of blood, even 20 c.c., will sometimes suffice, whereas in the treatment of acute anæmia due to hæmorrhage, the blood lost should be replaced as fully as possible, with due regard to the danger of dilating an enfeebled heart.

But great uncertainty exists as to the optimum dosage in the treatment of chronic anæmias. There is a tendency now to the belief that large single transfusions are less beneficial, so far at least as stimulation of the bone-marrow is concerned, than are small or moderate repeated transfusions, and I notice that the results which I have obtained in cases of pernicious anæmia with 250 c.c. doses compare favourably, even after a single transfusion, with those reported in America after such massive doses as 1,000 c.c. or more. Careful and prolonged trial of different doses administered at various intervals is necessary before more can be said as to the best routine to employ, but the following case is interesting as illustrating the apparent value of very small doses.

Case V.—A. P., aged 38, was admitted to the Metropolitan Hospital on May 14, 1914, under the care of Dr. Langdon Brown, suffering from hæmorrhage from the nose and gums, extensive purpura, spontaneous bruising and hæmaturia. No cause for the disease could be discovered. The symptoms were of sudden onset, and the epistaxis and bleeding from the gums persisted steadily after admission and in spite of treatment. After two months the patient's blood was reduced to under 500,000 red cells per cubic millimetre, and 12 per cent. hæmoglobin. At this stage small injections of blood were commenced, 20 c.c. being administered by syringe at intervals as shown on the chart. The course of the anæmia will be seen: progress was interrupted by a recurrence of spontaneous hæmorrhage, by an attack of influenza and by a profuse hæmorrhage after extraction of several teeth, but a year later the patient returned to work with a red cell count of 4,700,000 per cubic millimetre and 95 per cent. hæmoglobin. He has remained at work ever since, and his general health is good but the blood, although it contains 100 per cent. of hæmoglobin, shows at present only 3,500,000 red cells per cubic millimetre.

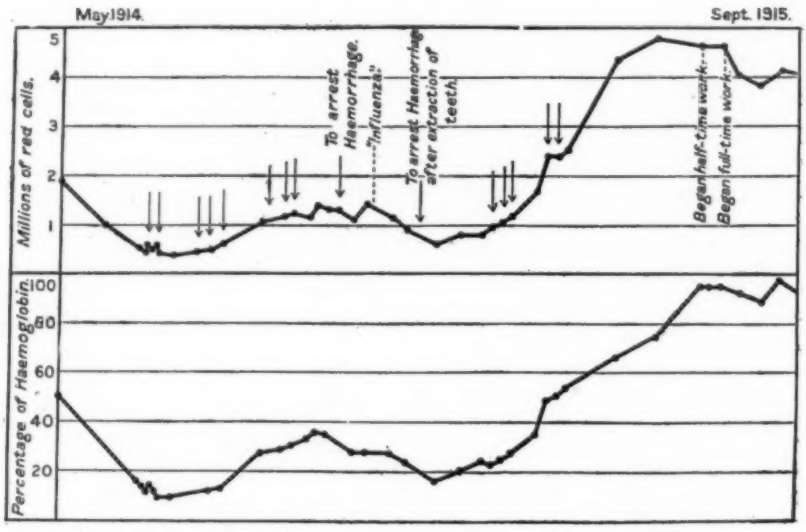

CHART IV.

At each of the points indicated by an arrow 20 c.c. of normal blood were injected intravenously.

METHODS OF TRANSFUSION.

The first consideration is to prevent coagulation of the transfused blood, the second to determine the amount which passes from donor to patient. The methods which have been employed may be classified as follows:—

(1) *Direct.*—By anastomosis of vessels, with or without a cannula.

(2) *Indirect.*—(i) By syringe: (*a*) syringe transferred from needle in vein of donor to needle in vein of recipient; (*b*) syringe with two-way tap leading to veins of donor and recipient. (ii) By use of a receiver: (*a*) with paraffin lining; (*b*) with addition of an anti-coagulant.

I have not attempted vascular anastomosis. The method is advantageous in avoiding exposure of the transfused blood, but it requires great surgical skill and the damage done to the blood-vessels employed is a serious drawback so far as the donor is concerned, while

it may prove disadvantageous to the patient if the operation has to be several times repeated. And further, no exact determination of the amount of blood transfused is possible.

The syringe method, popularized by Lindeman, requires two skilled operators, several other assistants, and a large number of expensive syringes which are easily broken. But given these various requirements the method is a good one.

The mechanical difficulties in connexion with the two-way tap are very great. I have had several taps made but finally discarded them in favour of a method employing a receiver.

The paraffin-lined apparatus of Satterlee and Hooker requires elaborate precautions and coagulation is very liable to occur, whereas the use of an anticoagulant renders transfusion a simple matter.

Satterlee and Hooker have employed hirudin to prevent coagulation with satisfactory results, but it is difficult to guarantee the absence of toxic substances in the hirudin and serious symptoms have occurred in some of the later cases in which it has been employed.

Two years ago Weil published his experiments on the transfusion of exsanguinated animals with citrated blood, and since then I have employed sodium citrate in twenty-nine transfusions. At the same time it has been very extensively used in America and its convenience is generally recognized, although some workers assert that it has a toxic action when used in adequate doses. All the clinical as well as the experimental evidence is opposed to this assertion. Weil recommended a 10 per cent. solution of the citrate, which is sufficiently nearly isotonic with the blood to avoid causing hæmolysis, and one part of this almost always prevents coagulation when mixed with ten parts of blood. Smaller quantities of citrate will often suffice, but a higher proportion has no disadvantages so far as I can determine. Salant and Wise have shown that the toxic dose of sodium citrate in dogs and cats is far greater than this, and Weil states that the coagulation time of the recipient is actually lowered by transfusion of citrated blood.

It may be questioned whether the citrate, by merely inhibiting the final stage in the process of coagulation, masks the preliminary stages which have in themselves rendered the blood toxic, but even if this is so the dangers involved are not great and the value of the transfused blood is not evidently diminished.

The apparatus which I employ sucks the blood from the vein of the donor into a glass receiver, while a graduated stream of 10 per cent.

solution of sodium citrate mingles with the blood as close as possible to its exit from the vein. When the required amount of blood has been withdrawn suction is stopped, the stream of citrate solution is cut off, the needle is removed from the vein of the donor and introduced into the vein of the patient, and the blood is gently pumped from the receiver. By this method the time employed in withdrawing and transferring 250 c.c. of blood is usually about fifteen minutes.

Summary.

(1) Transfusion has been successfully employed in the treatment of various kinds of anæmia and in the arrest of spontaneous hæmorrhage. It has also yielded promising results in cases of serious infection and in certain toxæmias.

(2) The ultimate prognosis in cases of anæmia depends upon the power of reaction in the bone-marrow and this can only be adequately determined by observing the results of treatment.

(3) In cases of pernicious anæmia both increased red cell production and diminished red cell destruction may result from transfusion.

(4) In cases of pernicious anæmia the age of the patient, the duration of the disease, and the condition of the bone-marrow as indicated in the peripheral blood, have hitherto proved to be the best guides to the progress subsequent to transfusion.

(5) The optimum dosage for transfusion is not yet determined, but it is probable that moderate repeated doses are preferable to large single doses in the treatment of chronic anæmias. Very small doses may sometimes be of value.

(6) The donor should be a healthy adult with negative Wassermann reaction. The serum of the donor should not agglutinate the corpuscles of the patient, and the serum of the patient should not agglutinate the corpuscles of the donor. Agglutinins should be excluded by tests done immediately before the transfusion, and a single examination is not sufficient to establish the compatibility of two bloods on all future occasions. If agglutinins are absent, hæmolysins will also be absent.

(7) If there be great urgency, and testing of the blood of patient and donor be impracticable, a small preliminary transfusion should be done half an hour before the main mass of blood is transfused so that gross incompatibility may be recognized in time.

(8) Febrile reactions occur after about 25 per cent. of transfusions, even though the bloods of donor and patient have been proved to be "compatible." Rigors occur in about 10 per cent. of the cases.

(9) It may prove desirable to investigate the bloods of donor and patient with regard to factors of which we as yet know nothing, not merely for the sake of avoiding accidents, but also to determine whether a given donor is likely to afford the maximum of benefit in a particular case.

(10) The indirect method of transfusion, employing a glass receiver and sufficient sodium citrate to prevent coagulation of the transfused blood, is simple and involves no special dangers.

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DISCUSSION.

Dr. O. LEYTON: Dr. Stansfeld told us that in one case in which subsequent examination showed that the bloods were not well matched, the transfusion was followed by cramp and vomiting. Some years ago I transfused a patient suffering from severe pernicious anæmia with the blood from the only case of erythræmia available at the time. After an ounce or two of blood had been injected the patient complained of very severe pains all over him. The transfusion was stopped and the pains subsided within a few minutes. The patient improved to so great an extent that he asked for a second transfusion. On this occasion between 3 and 4 oz. were injected, which led not only to severe pains but also to nausea, vomiting and retching, accompanied by a tachycardia which gave rise to grave anxiety. Recovery was complete two hours after the transfusion. My impression is that the condition is similar to caisson disease and is produced by clumps of red cells temporarily obstructing vessels of the central nervous system. During the last three months I have given nineteen massive transfusions to a patient suffering from intense pernicious anæmia whom many at one time considered moribund. The usual remedies had proved of no avail. I found that in this case large quantities of blood, not less than a pint, seemed to do at least four times as much good as half that quantity. The multiple syringe method was adopted, to begin with; difficulty arose due to the blood clotting in the needle in the donor's vein. This was overcome by using a double needle; the inner one could be removed and replaced by a clean one without disturbing the outer. The same kind of needle was used in the patient; by these the trouble of clotting was completely removed and on many occasions, with the skilled assistance of Dr. Panton, I have transferred 640 c.c. of blood from the donor to the patient within half an hour. My experience has been similar to that of Dr. Stansfeld in that even when the bloods matched perfectly, rigors and pyrexia have followed. It occurred to me that the symptoms were in some ways similar to those in anaphylaxis and therefore, remembering the observations of Professor and Dr. Helen Leyton, I injected hyoscine methyl bromide, with a very small dose of morphine, into the patient half an hour before the transfusion. The results have been most satisfactory. Shortly after the transfusions the colour of the urine has been even darker than usual, suggesting that the foreign red cells were rapidly destroyed. Evidence of this was forthcoming from red cell counts. The day following the transfusion there was a great increase which often disappeared in a few days. This led to my giving the transfusions at very short intervals. The red cells gradually rose to 3,000,000 and the hæmoglobin to 60 per cent. and then I thought it wise to have the spleen removed. The patient is now convalescent from the operation and has lost

completely the yellow colour so characteristic of pernicious anæmia. The other conditions in which Dr. Stansfeld has found transfusion beneficial suggest a wide future for the treatment, and I have every expectation and hope that a new and satisfactory method of assisting recovery has been brought to the notice of the profession by Dr. Stansfeld.

Dr. F. PARKES WEBER: I should like to ask Dr. Stansfeld whether he has tried the effect of intramuscular injections of the patient's own defibrinated blood. I mean to say, whether he has withdrawn some blood from one of the patient's veins, defibrinated it, and injected the defibrinated blood back again into the patient (into the gluteal muscles). I do not refer to cases of severe anæmia, but I understand that such a method of treatment has been tried in cases of very troublesome pruriginous, purpuric and erythematous eruptions, &c., and that some good results have been claimed.

Dr. LANGMEAD: Has Dr. Stansfeld any experience of the use of this method of treatment in the hæmorrhagic disease of the newly born? The condition is one which has been treated by transfusion, by injection of blood serum, and by intramuscular injections of blood and the results in each of these forms of treatment have been very encouraging. At one time regarded as an almost fatal condition, its prognosis has been quite altered by these means, and if the cases are treated early enough, the infants' lives are usually saved. It would appear that in such cases a deficiency of bodies necessary for sufficient coagulation is made up.

Dr. STANSFELD (in reply): With regard to the wide application of transfusion in the treatment of disease, it should be remembered that the blood is an exceedingly complex substance, and that different elements in it may be responsible for benefiting very different conditions. The scope of transfusion is not yet determined but so far as the anæmias are concerned, transfusion, by stimulating the bone-marrow, might be of value in very varying conditions. It is certainly not to be looked upon, however, as a panacea for all anæmias. I do not think that the cases of pernicious anæmia which I have treated were essentially different from those cases which are amenable to ordinary methods of treatment. They differed rather in degree and apparently unfavourable cases were chosen, partly because it was thought they would afford a better test of the method, partly because of the difficulty experienced in obtaining permission to try it. I am indebted to Dr. Langdon Brown for the first clinical material placed at my disposal. The difficulty experienced in procuring donors accounts for the fact that I began by using comparatively small doses, and I have thought it well not to vary the dose in so small a number of cases. I have no personal experience of such doses as Dr. Leyton has used. I shall be very interested in trying his method of preventing rigors. It was melæna neonatorum, to which Dr. Langmead has referred,

which I had in my mind when I pointed out the advantage of knowing that infants may be safely transfused from their mothers without preliminary testing of the bloods. If the child has lost much blood the mother's corpuscles will be of value, but if the condition is seen very early an intramuscular injection of blood may suffice. This is very easily given and causes very little pain. I have given as much as 250 c.c. of blood into the buttocks of an adult on a single occasion without causing more than a few minutes' pain. In reply to Dr. Weber, I have tried autoserotherapy in one case of urticaria without benefit. In one very troublesome case of urticaria transfusion apparently brought about a brilliant result, but my results with blood and serum therapy in cases of skin disease have been inconstant and on the whole disappointing.

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

J. Y. W. MacALISTER

UNDER THE DIRECTION OF

THE EDITORIAL COMMITTEE

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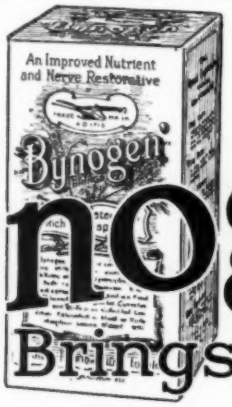

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